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FURTHER OBSERVATIONS ON NEUROLOGICAL ABNORMALITIES IN MENTAL DEFECTIVES

BY

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IN 1933 we published a survey of the neurological findings in 500 defectives, in which paper, as in the present one, the object was to discover possible variations from normal in reflexes and other neurological phenomena in those whose nervous systems might be developmentally imperfect. From both these studies it transpires that there is a considerable variation from normal in these findings, quite apart from the occurrence of clinically recognizable syndromes. In the present paper subjective factors such as observations on sensations have been omitted, since they are open to the criticism that the information obtained from defectives may not be dependable. The present investigation has therefore been confined to purely objective manifestations of the motor performance and reflex findings in mental defectives, with the object of determining the functional capacity of their motor apparatus.

The material consisted of 336 unselected mental defectives of both sexes and all ages. These were divided into the following grades : feeble-minded, 85 (25 per cent.) ; imbeciles, 177 (53 per cent.) ; and idiots 74 (22 per cent.).

In observing a group of mental defectives one of the most striking peculiarities to be noticed is the poor posture and apparent general slackness of muscular tone which these individuals present. That this may be rather an expression of poor general co-ordination than of any real alteration of muscle tone is suggested by the observation that when instructed in drill and deportment according to their capacities, many defectives assume a general bodily posture which is indistinguishable from that of the normal individual.

In order to test this general impression an attempt was made to determine if many of these persons showed a disturbance of muscular tone which could be described as pathological. For this purpose tone was classified as: (1) normal, (2) plastic, (3) spastic, (4) spastic with contractures, (5) contracture without spasticity, (6) catatonic, and (7) flaccid. Table I shows the results of this investigation.

TABLE I

	TOTALS	NORMAL	PLASTIC	SPASTIC	SPASTIC + CONTRACTURES	CONTRACTURES — SPASTICITY	CATATONIC	FLACCID
Feeble-minded	85	78 (92%)	1 (1%)	1 (1%)	1 (1%)	3 (3%)	1 (1%)	0
Imbeciles	177	141 (80%)	6 (3%)	11 (6%)	8 (5%)	5 (3%)	1 (1%)	5 (3%)
Idiots ..	74	48 (65%)	1 (1%)	14 (18%)	1 (1%)	0	0	10 (14%)
Total ..	336	267 (79%)	8 (2%)	26 (8%)	10 (3%)	8 (2%)	2 (1%)	15 (4%)

The number of cases presenting abnormal muscle tone is thus seen to be relatively small, viz. 21 per cent., and of these 12 per cent. of the whole group is made up of the spastic cases of hemiplegia, tetraplegia, diplegia, and paraplegia. Thus only 9 per cent. of those not presenting clinical syndromes show abnormalities of muscle tone.

The association of variation in tone with signs of pyramidal system dysfunction as exemplified by the following criteria was investigated: exaggeration of the biceps, wrist, or ankle-jerks; knee-jerks with positive Strumpell's sign (i.e. obtained off tibia); patellar or ankle clonus; dorsiflexion of great toe in response to stroking of front of tibia; extensor plantar response.

TABLE II

	TOTALS	NORMAL	PLASTIC	SPASTIC	SPASTIC + CONTRACTURES	CONTRACTURES — SPASTICITY	CATATONIC	FLACCID
Feeble-minded	15	12 (80%)	0	0	1 (7%)	1 (7%)	1 (7%)	0
Imbeciles	72	49 (68%)	3 (4%)	9 (12%)	7 (10%)	4 (6%)	0	0
Idiots ..	30	17 (57%)	1 (3%)	10 (33%)	1 (3%)	0	0	1 (3%)

From these tables it is obvious that alterations in tone may occur without definite evidence of permanent recognizable lesions in the nervous system. It is of interest that in the group of the feeble-minded, abnormalities of tone are infrequent, while in the group of idiots such abnormalities are common. It is possible that such variations of tone in some cases depend more on a diffuse cortical inferiority with resultant defect of co-ordination, integration and inhibition than on localized lesions.

Disturbances of general co-ordination were obviously prevalent, and a study of these was next undertaken. In the previous study the prevalence of

choreiform movements amongst mental defectives had been noted in 25 per cent. of a total of 500. In the present series evidence of hyperkinesis was found as shown in Table III. This is clearly more prevalent than in normal individuals of the same age, and is a further indication that in mental defectives cortical control is likely to be poor. As to the choreiform movements, none were of a gross nature, resembling rather what is found in a mild or residual case of Sydenham's chorea. No correlation with reflex abnormalities was found. There was, however, a tendency for hyperkinesis to be associated more frequently with hypotonia than with hypertonia.

TABLE III

	TOTALS	TREMOR	CHOREA	ATHETOSIS	TICS	TOTAL HYPERKINESIS
Feeble-minded ..	85	10 (12%)	34 (40%)	0	0	44 (52%)
Imbeciles	177	17 (10%)	92 (52%)	15 (8%)	1 (0·5%)	125 (70·5%)
Idiots	74	4 (5%)	32 (43%)	3 (4%)	0	39 (52%)

The " cuisse-tronc " sign of Babinski, which depends upon faulty synergia of the back muscles, especially of the glutei, as shown when the subject lying on his back raises his trunk off the ground without the assistance of his hands, was also studied. A positive " cuisse-tronc " sign was indicated by the raising of the legs off the ground on sitting up. Babinski suggested that this occurred especially in the presence of lesions of the pyramidal tract. The results are shown in Table IV.

TABLE IV

	TOTAL	POSITIVE " CUISSE-TRONC " SIGN
Feeble-minded ..	85	19 (22%)
Imbeciles	177	55 (31%)
Idiots..	74	47 (63%)

This type of inco-ordination was more manifest the lower the grade of defective, which may be explicable by the relatively low efficiency of their nervous system as a whole. Again, it was found that this sign of inco-ordination was by no means confined to cases in which the pyramidal tracts or cells were specially involved. It occurred in an appreciable number of cases whose reflex anomalies suggested a definite muscular hypotonia. The presence of this sign may therefore be taken as a manifestation of a general inco-ordination of the neuro-muscular apparatus rather than of any single localized lesion in the nervous system.

The following tests of cerebellar function (Tilney and Riley, 1928) were then made the subject of study : (1) disturbance of walking and turning ; (2) Romberg's test ; (3) past pointing test (hands and feet) ; (4) diadokokinesia ; (5) rebound test ; (6) leg raising. It must be realized that too much reliance could not be placed on results of such tests in defectives, as some

individuals had difficulty in understanding instructions. Taking the total errors of co-ordination found in relation to these tests Table V indicates the findings. It is clear that there is an inferiority on the part of low-grade subjects in their ability to perform such tests. When this is correlated with reflex variations results are obtained as shown in Table VI.

TABLE V

	TOTAL	CASES SHOWING SOME FAILURE IN CEREBELLAR TESTS
Feeble-minded ..	85	35 (41%)
Imbeciles	177	115 (65%)
Idiots..	74	70 (95%)

TABLE VI

	TOTAL	WITH PYRAMIDAL SIGNS	WITH DIMINISHED REFLEXES	WITH NORMAL REFLEXES
Feeble-minded ..	35	7 (20%)	5 (14%)	23 (66%)
Imbeciles	115	50 (43%)	19 (17%)	46 (40%)
Idiots	70	29 (41%)	14 (20%)	27 (9%)

It will be seen, therefore, that more than half of the subjects with signs usually attributed to cerebellar dysfunction also show reflex anomalies indicative of disturbance of function in the nervous system either central or peripheral. It is difficult to be sure where the failure lies which determines the incoordination. It seems probable that a general irregular failure of the influences impinging on the final motor pathway is present, rather than specific cerebellar dysfunction. This is in accordance with the observed fact that cytoarchitectonic abnormalities in the defective are widespread ; and that the most significant change is an irregular arrangement of cells. It is therefore improbable that the functional co-ordination of cortical effector cells, corpus striatum, red nucleus, cerebellum, vestibular apparatus, and spinal motor centres is really effectual in these cases. A gross defect in function on both sides of the arc is suggested by our previous observations of remarkably poor performance tests in spatial discrimination.

In the previous study we had included reflex anomalies in our general survey of neurological behaviour. In the present series the following reflexes were systematically examined : biceps-jerk, wrist-jerk, knee-jerk, ankle-jerk, plantar reflex, Oppenheim's reflex, and the presence of knee and ankle clonus.

Dividing the cases into (1) those who showed reflex changes suggesting anomalies in the pyramidal system, (2) those with definitely diminished reflexes, and (3) those with reflex responses within the normal range, the distribution is found in Table VII. As might be expected, the lower the grade the less likely were the reflexes to be normal. This was partly accounted for by those cases in the group showing definite clinical syndromes. It is clear, however, that many defectives show reflex anomalies which have no apparent relation to

established clinical syndromes. The question arose as to whether these reflex changes were or were not constant. Eighty unselected cases which had previously shown signs indicative of pyramidal system defect were therefore re-examined after an interval of at least 12 months. Twenty (25 per cent) showed definitely recognizable clinical syndromes of diplegia, hemiplegia, or paraplegia, while 60 (75 per cent.) manifested no such syndromes. In only three cases (3·75 per cent.) had all signs of such defect passed away. In 24 cases (30 per cent.) the findings were in all respects the same. In eight of these 24 there were recognizable syndromes, thus there was no evidence that these conditions had progressed. In the other 16 cases there was no evidence that any such syndromes were present. In 26 cases (32·5 per cent.) the manifestations of pyramidal defect had become more marked. Of these only eight showed definitely recognizable syndromes. It would appear to be somewhat rash to conclude that symptomatically the pathological condition, except in two of the syndrome cases, had definitely advanced. In the remaining 27 cases (33·25 per cent.) the pyramidal signs were altered in their distribution, but so far as could be observed not in any significant way. Of these only four suffered from a definitely recognizable syndrome, and 24 merely showed further variations in the already inconsistent state of their reflex record.

TABLE VII

	TOTAL	WITH PYRAMIDAL SIGNS	WITH DIMINISHED REFLEXES	WITH NORMAL REFLEXES
Feeble-minded ..	85	15 (18%)	17 (20%)	32 (62%)
Imbeciles	177	72 (41%)	28 (16%)	100 (43%)
Idiots	74	30 (41%)	16 (22%)	46 (33%)
All grades	336	117 (35%)	61 (18%)	178 (47%)

To amplify the neurological findings in mental defectives the organic reflexes were investigated in a similar group of low-grade patients. This group comprised 500 defectives who had a mental age between 1½ and 6 years, as estimated by the Merrill-Palmer test. It will be seen that in the subjects constantly incontinent the chronological age was higher and the mental age lower than that for the whole of the series. Constant double incontinence is seen to be much more frequent in males than in females, and this difference is statistically highly significant, for it exceeds eight times the probable error. The association between a lower mental age and double incontinence is not absolute. Nevertheless on the average the mental age of the subjects with double incontinence is definitely lower. These figures suggest that, amongst defectives, the lower the mental age the greater the tendency to incontinence and that incontinence amongst defectives is commoner in males than females.

Though not established by the present enquiry, it would appear probable that voluntary control over the evacuation of urine and fæces is acquired later by defectives than by normal children. In the cases studied none of the defectives, with one exception, was below the age of 7 years. Above this age there was no reduction in the proportion of constantly incontinent cases as chronological age increased, thus showing that these patients do not " grow

out of it." The low mental age of these defectives, coupled with other available information, suggests that in them cortical development, though much below normal, has in the majority been at least sufficient to enable control to be established over unchecked spinal activity.

TABLE VIII

TOTAL	PERCENTAGE INCONTINENTS	PERCENTAGE CONSTANT DOUBLE INCONTINENCE	MEAN CHRONOLOGICAL AGE	AVERAGE MENTAL AGE (MERRILL PALMER-TEST)	CHRONOLOGICAL AGE OF 62. CONSTANTLY INCONTINENT 52 M. ; 10 F.	MENTAL AGE OF 62. CONSTANTLY INCONTINENT 52 M. ; 10 F.
Males .. 264	48·7	19·8	18·5 yrs.	3·6	19·2	2·8
Females 236	27·5	4·2	20·6 yrs.	3·8	21·5	2·8

In those with double incontinence it is significant that the mental age is low. It is possible that in defectives the sacral segments of the spinal cord may show structural defects. If this be so disturbances might be expected in both the somatic and autonomic reflexes of the parts concerned. To test this an examination of the bulbo-cavernosus and superficial anal somatic reflexes was carried out in 51 of the 52 doubly incontinent males. As a control 51 continent males of as far as possible equivalent mental ages were examined in the same way. The results of this examination are given in Table IX.

TABLE IX

	DOUBLE INCONTINENCE		CONTROLS	
	No.	%	No.	%
Bulbo-cavernosis and anal reflexes present	7	14	22	43
Bulbo-cavernous only	9	18	9	18
Anal only	0	—	4	8
Both reflexes absent	35	69	16	31
	51		51	

There are individuals in both groups with absent sacral somatic reflexes and also individuals in the two groups in whom the reflexes are present. Nevertheless the proportion of individuals in the control group with normal reflexes is significantly greater than the proportion in the incontinent group.

An attempt was made to determine the relation, if any, between the state of these somatic reflexes and the chronological and mental ages of the patients. In order to make this analysis it was necessary to know whether chronological and mental ages are correlated in these two groups. It was found that there was no such correlation, and therefore it was legitimate to examine separately the relation between the state of the reflexes and chronological and mental age respectively. It was found that the mental age of the control group exceeded that of the group with incontinence. This was unavoidable, as there were not enough individuals of low mental age in the former group to make the correspondence exact. In the group with incontinence there was no evidence of any association between the state of the reflexes and either chronological or mental age. In the control group, however, individuals with absent somatic reflexes had a rather lower mental age and a definitely lower chronological age.

The following deductions may be drawn from the results of this part of the examination : (1) The majority of the group with incontinence have some structural abnormality in the sacral segments of the cord which within the age limits investigated is of a permanent character. (2) A smaller proportion of the control group display a similar abnormality of somatic reflexes and the figures suggest that the reflexes may sometimes appear relatively late in life in individuals. (3) In the control group there appears to be some association between absence of reflexes and a specially low mental age. In view of these findings it would appear that in defectives who are incontinent there may be in addition to the cerebral defect a permanent abnormality in the proper development of the sacral segments and that this may stand in causal relationship to the incontinence.

In defectives in whom the sacral segments are efficiently developed so as to allow for proper organization of the reflexes, permanent incontinence is only met with in the presence of markedly imperfect cortical function, while in those cases in which the sacral development is interfered with, relatively higher grades of generalized cortical deficiency may still exhibit permanent complete incontinence.

Summary

An investigation of reflex abnormalities has been made in a group of 336 unselected institutional mental defectives.

Abnormal neurological signs were found more frequently in the lower-grade patients. In the total group abnormal variations in muscle tone were present in 21 per cent., hyperkinesis in 61 per cent., the " cuisse-tronc " sign of Babinski in 36 per cent., inco-ordination in 65 per cent., and signs of pyramidal tract dysfunction in 35 per cent. of cases. The association of these various abnormalities among themselves has been examined.

A re-examination of 80 unselected cases which had shown signs of pyramidal system defect was made after an interval of at least one year. In approximately one-third the signs had become more marked, in one-third the condition appeared stationary, in the remainder the abnormal neurological signs had altered in distribution while in only three cases had all evidence of defect passed away.

An investigation of some of the factors influencing incontinence of urine and fæces has been made in a group of 500 mental defectives. Constant incontinence was found to be associated with low mental age and to be much more frequent in males than in females. In defectives with incontinence the bulbo-cavernosus and superficial anal reflexes were more often absent than in a control group of equivalent mental age. It was concluded that in addition to poor cortical control a permanent abnormality of the sacral spinal segments is often a factor in the establishment of incontinence in mental defectives.

REFERENCES

Gordon, R. G., Norman, R. M., and Berry, R. J. A. (1933). *J. Neurol. Psychopath.*, **14**, 97.
Gordon, R. G., and Norman, R. M. (1932). *Brit. J. Psychol., I* and *II.*
Tilney, F., and Riley, H. A. (1923). " The Form and Function of the Central Nervous System," 2nd edition, p. 478.

THE ELECTRO-ENCEPHALOGRAM IN CONVULSIONS INDUCED BY CARDIAZOL

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SEIZURES induced by cardiazol (pentamethylene tetrazol) are not identical with *Grand Mal*, but the clinical resemblance is sufficiently close to justify their study by electro-encephalography. Gibbs, Gibbs, and Lennox (1937) have shown conclusively that a genuine epileptic fit is accompanied by a rhythmic, high-potential electrical discharge from the cortex, and Golla, Graham, and Walter (1937) have produced evidence that the majority of young idiopathic epileptics exhibit a focal resting discharge which, by analogy with the slow waves found in the neighbourhood of cerebral tumours (Walter, 1937) have been called " delta foci." Moreover, when an epileptic patient with such a focus has a fit during electro-encephalography, the large waves characteristic of the fit appear first in or near the delta focus and spread thence to the rest of the cortex. Now in the majority of epileptics who show such a focus and complain of frequent major fits, the focus is in the region of the superior frontal gyrus, sometimes on one side only, sometimes on both. This area corresponds roughly with the " Area 6aβ " of Vogt, to which Foerster (1936) and others have attributed an " epileptogenic " function. The anatomy and physiology of this part of the cortex and its relation to other parts are not yet sufficiently clear to warrant the adoption of any definite hypothesis, but there is evidence from several sources that this part of the frontal lobe is concerned in some way with the development of convulsive seizures. The possibility of taking records during a series of controlled convulsions induced in the course of treatment of schizophrenic patients at Bexley Hospital seemed an excellent opportunity for collecting information on this matter.

The equipment was the same as that used by one of us for the study of cerebral tumours (Walter, 1937). It consists of a three-channel amplifier and triple cathode-ray oscillograph unit ; the amplifiers have balanced input stages and are therefore entirely independent of one another, and the oscillographs have screens with a long after-glow which makes the observation of slow changes easy, if it is not desired to take records. With this apparatus the place of origin of a cortical discharge can be located to within a centimetre or

so by observing the phase relations of the potential changes in the three leads. A more detailed explanation of this technique is given in the paper referred to above.

The first difficulty encountered was that some of the patients, by reason of their condition, were unwilling to co-operate and violently opposed the fitting of the cap and electrodes. This meant that many records were spoiled by the movements of the patients or the restraining efforts of the assistants. The second obstacle was that during the intravenous injection of the cardiazol the patient's arm must be firmly held, and at the beginning of the convulsion a gag must be placed in the mouth, so that even if there is no struggling, the patient must be touched twice at least, which, as is well known to those who work with amplifiers, is liable to introduce large artefacts into the record, particularly since the patient is not at earth potential when balanced input amplifiers are used. The size of these artefacts was reduced by the nurses wearing rubber gloves when touching the patient.

Sixteen patients were under treatment during the course of the experiments, and of these 14 were found sufficiently co-operative. Observations were made during 32 injections and satisfactory records were obtained from 12 typical convulsions. Artefacts from the sources mentioned above spoiled 13 of the experiments and in five cases the patient failed to have a fit.

The results obtained were the same in all the experiments in which the effect of the injection was comparable. They may be divided into two groups : first, those cases in which the injection failed to induce a convulsion, and secondly, those in which the injection was followed by a typical general convulsion. In all cases records were taken before the injection, first with high amplification, in order to observe the character of the resting electro-encephalogram, and then with the much lower amplification, which was used later to show the very large potentials which were expected to accompany the fit. When the needle was in the vein, continuous recording or observation was started and continued until either the convulsion was over or it was clear that the patient was not going to have one.

The patient whose electro-encephalogram is here illustrated was a married woman of 26, suffering from a schizophrenic illness of 2 years' duration. On admission she was depressed and apprehensive, introverted, and so preoccupied with morbid ideas as to be practically inaccessible to the usual inquiries. Hallucinations and schizoid grimacings and mannerisms were always in evidence, whilst later typically hebephrenic symptoms were superadded. Her physical condition was good and there was no evidence of neurological lesion.

On 26th June, 1937, a course of cardiazol was started, with the result that after five fits she was in a state of very fair remission and was perfectly co-operative as regards electro-encephalography.

Description of Fits induced by Cardiazol

The fits are produced by rapid, intravenous injection of a 10 per cent. ardiazol solution into one of the antecubital veins. They are in the nature of

typical, strong, generalized epileptic convulsions. The first sign, which is usually a short cough, blepharospasm, or mouth twitching, occurs from 3 to 15 seconds after the end of the injection. Large, myoclonic jerkings of head, limbs, and body follow (8 to 20 seconds after the injection) and usually last from 2 to 10 seconds. The tonic phase usually begins 10 to 30 seconds after the injection and is always very severe. Its duration varies from 5 to 20 seconds, when it merges into the clonic phase, which starts approximately 18 to 40 seconds after the injection and generally lasts from 25 to 55 seconds. Flaccidity, sometimes interrupted by an occasional generalized myoclonic jerking, usually appears from 55 to 90 seconds after the injection. The pupils are nearly always dilated throughout the fit and the eyes often show extreme lateral deviation, to either side even in the same case. Other frequent accompanying features are goose-skin, micturition, and spontaneous extensor plantar sign.

In the case illustrated nine fits were induced between 26th June and 23rd July, 1937. The first three fits followed a dose of 0·5 gm. cardiazol (5 c.c. of the 10 per cent. solution), the fourth fit required 0·6 gm., the fifth fit 0·7 gm., and the last four fits, 0·8 gm. On 12th and 13th July, 1937, electro-encephalograms were taken. On 12th July, 0·7 gm. cardiazol was administered without producing a major fit. Except for a blank, staring expression and widening pupils, no signs appeared until 20 seconds after the end of the injection, when prolonged blepharospasm occurred. There were no convulsive movements, but for about 2 minutes short, quick, blowing respirations were in evidence. During this period the patient was semi-conscious and mentally inaccessible.

On the following day the injection of 0·8 gm. cardiazol produced a typical fit, ushered in by a short cough 5 seconds after the end of the injection, and followed immediately by blepharospasm (7 seconds). The tonic phase occurred at 17 seconds, clonus at 35 seconds, and the patient became flaccid at 50 seconds. There was deviation of the eyes upwards and to the right.

The experiments in which no fit followed the injection are of some interest. A typical observation was as follows. For a few seconds following the injection no change is seen in the electro-encephalogram, then small waves appear in all leads (Fig. 1). These have a frequency of about 6 per second and involve all areas of the cortex. They increase gradually in size for about a minute, and remain for some hours. During the first few minutes the patient is confused and may exhibit behaviour of the " epileptic equivalent " type, whilst later there may be feelings of uneasiness and distress lasting several hours and the symptoms of the psychosis may be more pronounced than before the injection. The electro-encephalogram does not resemble in any way that found in cases of *Petit Mal*, for this condition is characterized by a focal discharge at a frequency of about 3 per second, while the sub-convulsive doses of cardiazol produce a general discharge at about twice that frequency. A more detailed description of these cases is contained in a paper by one of us (Cook, 1938) on the clinical aspects of the treatment.

When the injection induces a convulsion, the electro-encephalogram starts in the same way after a latency of a few seconds (Fig. 2). The waves appear in all areas, but grow more rapidly and have a slightly lower frequency ; at about

15 seconds after the injection there is an abrupt increase in size and drop in frequency of the waves in the frontal region. Sometimes this occurs first on the left, sometimes on the right, sometimes it appears almost simultaneously on both sides. At this moment the tonic stage of the convulsion starts. The area involved by large slow waves increases and spreads over the greater part of the cortex, then, as the fit passes into the clonic stage, the discharge becomes more staccato in rhythm with the jerks, then suddenly ceases from 1 to 1½ minutes after the injection. The electro-encephalogram is now abnormally quiet, no waves of any sort being seen, and the patient is completely relaxed and flaccid. As consciousness returns the normal small random potentials reappear, and 30 minutes later neither patient nor electro-encephalogram shows any trace of the storm which has agitated them.

The striking feature of the records in these convulsions is that, although the diffuse discharge starts in the same way as when no fit follows, there is a sudden increase in potential and drop in frequency in one area. Records taken with electrodes suitably placed for localizing in the frontal lobes show that the area where this sudden outburst occurs is in the neighbourhood of the superior frontal gyrus on one side or the other. The rapidity of development of the seizure prevented any correlation being established between the side of the electrical change and the side on which the convulsion started. Clinically the fits do not suggest any focal origin in the usual sense, any more than does a typical major attack, but in every case in which a satisfactory record or observation was made, there was a definite focal discharge as a prelude to the convulsion, and the focus in every case was in approximately the same relative position as the resting delta foci of idiopathic epilepsy.

In order to account for this observation, it cannot be supposed that cardiazol has an entirely selective action on one part of the cortex, since small doses produce an abnormal discharge in all areas : it may be that when a certain concentration of cardiazol is reached, this frontal area becomes unstable and acts as a detonator for the rest of the cortex, which is itself already poisoned by the drug. It should be remembered that the electrical threshold for convulsive movements has been found to be lower in this area than in other parts of the brain. Another possibility is that this area, by reason of its internal anatomical connections, is the first to signal the onset of an abnormal condition in some sub-cortical centre. In any case, in view of the prevailing uncertainty as to the normal function and connections of this part of the central nervous system, the precise relation between the electro-encephalographic changes and the motor and psychic disturbances must remain, for the time being, a subject for speculation.

Summary

Electro-encephalograms have been taken from patients undergoing treatment by cardiazol injection for schizophrenia.

When an injection failed to induce a fit an abnormal discharge was found in

all areas of the cortex. This lasted for some hours and corresponded with a period of mental confusion and malaise.

When the injection was followed by a convulsion, the diffuse discharge was interrupted by a focal outburst coincident with the onset of the tonic spasm. This focus was always in the region of the superior frontal gyrus. When the fit was over the electro-encephalogram soon resumed its normal character.

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REFERENCES

Bumke, O., and Foerster, O. (1936). *Handbuch der Neurologie*, Springer, Berlin.
Cook, L. C. (1938). *Proc. roy. Soc. Med.* (in Press).
Gibbs, F. A., Gibbs, E. L., and Lennox, W. G. (1937). *Brain*, **60**, 377.
Golla, F. L., Graham, S., and Walter, W. G. (1937). *J. ment. Sci.*, **83**, 137.
Walter, W. G. (1937). *Proc. roy. Soc. Med.*, **30**, 579.

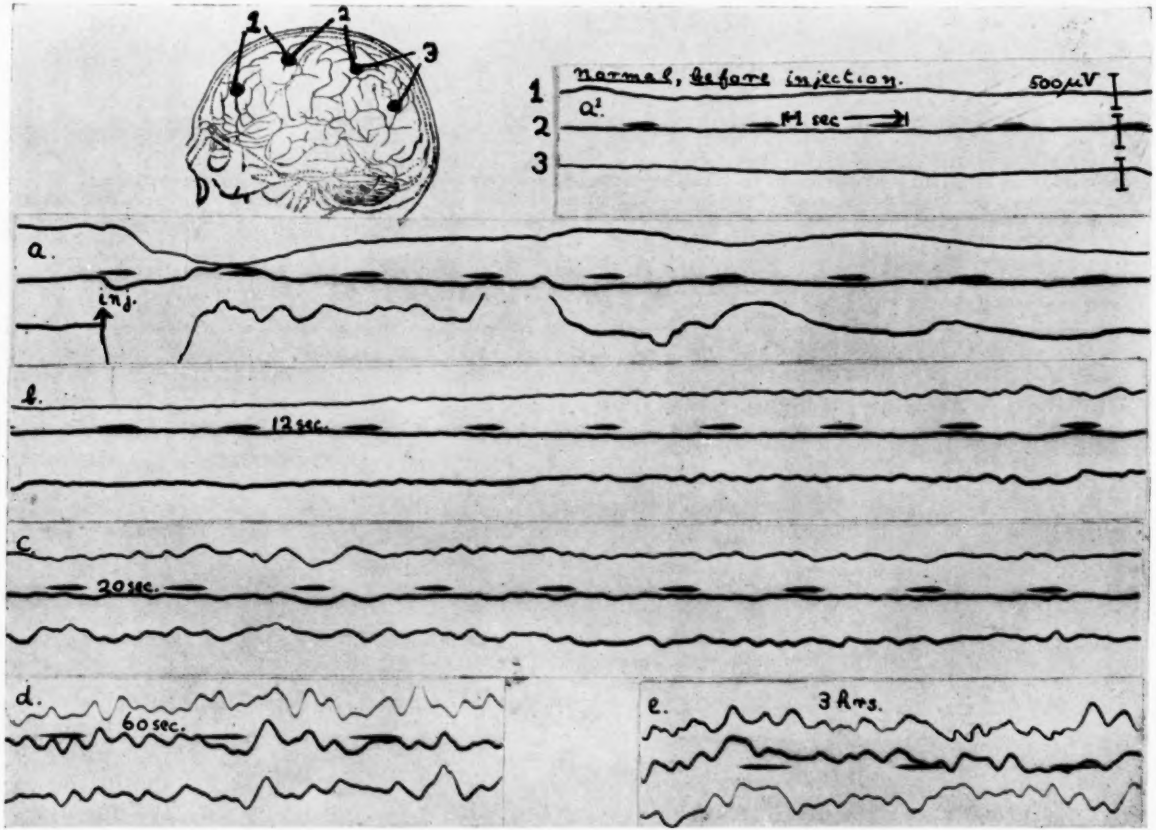

Fig. 1.—Electro-encephalograms taken on 12th July, 1937, typical of the effect of a sub-convulsant dose of cardiazol. Record *a*1 shows the time-scale and sensitivity, which are the same for Fig. 2. Records *a*, *b*, *c*, *d*, and *e* show the development and persistence of the slow diffuse delta-discharge.

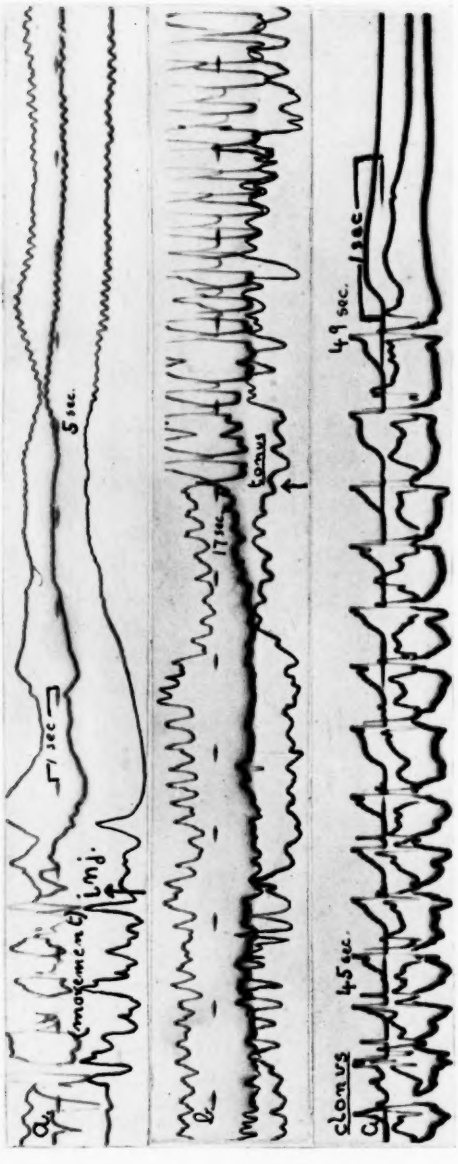

Fig. 2.—Electro-encephalograms taken on 13th July, 1937, showing the mode of development of the discharge associated with a cardiazol convulsion. Time-number, electrode arrangement, and sensitivity as in Fig. 1. Record *a* shows the injection which was accompanied by considerable movement, *b* shows the beginning of the tonic stage and *c* part of the clonic stage and the end of the convulsion.

THE RELATIONSHIP BETWEEN THE BLOOD PRESSURE AND THE TONIC REGULATION OF THE PIAL ARTERIES

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From the Medico-physiological Institute of the University of Copenhagen

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IN experiments on cats it was demonstrated that a decrease of blood pressure was followed by a dilatation of the pial arteries (Fog, 1934, 1937 ; Forbes, Nason, and Wortman, 1937) and that, conversely, a rise of arterial pressure caused a constriction of these vessels (Fog, 1938*a*). These vasomotor reactions were found to be independent of the procedures by which the fluctuations of pressure were produced. Stimulation of the sinus nerve or of the peripheral end of the cut vagus nerve or a diminution of blood volume had the same effect on the diameter of the pial arteries, provided that the same decrease in blood pressure occurred. Also the reactions during arterial hypertension were identical whether the rise in pressure was effected by the administration of adrenalin intravenously, stimulation of the splanchnic nerve, or increasing the blood volume. In this connection it is important to note that adrenalin was shown to have no direct chemical action upon the pial arteries of less than 50μ in diameter. On the other hand, vessels of larger than 100μ in diameter reacted to the hormone by distinct constriction, which, however, was not as great as that usually found in other vascular areas, e.g. the skin or the splanchnic area (Forbes and Wolff, 1928 ; Forbes, Finley, and Nason, 1933 ; Fog, 1934, 1938*b*).

It was concluded from these observations that the muscular tonus of the pial arteries was controlled by the intravascular pressure, hypertension causing augmentation and hypotension diminution of the tone. The regulation, however, has certain limits. Thus, a sudden change of blood pressure may at first cause a " passive " reaction : the vessels dilate when a rise of tension starts abruptly or narrow at the beginning of a phase of hypotension. After 1 or 2 minutes these changes are usually followed by an " active " constriction or, in the second case, dilatation. If the rise of blood pressure is more than 100 per cent. above the normal level, dilatation of the arteries will almost certainly follow ; and if the level of blood pressure at the commencement is below 50 mm. of mercury, active changes in the vessels do not occur.

It appears from the experiments mentioned that the pial precapillary arterioles maintain at the normal level of arterial tension a minimum diameter, which cannot be reduced markedly by fluctuations of the intravascular pressure. If the systemic pressure descends below 100 mm. of mercury there is a widening of the lumen proportional to the degree of hypotension ; during a subsequent rise to the normal level the artery will narrow to its original size. If the pressure is raised to 200–250 mm. of mercury there will not be any further constriction. The tone of the arterial muscular wall is sufficiently augmented just to prevent distension, though excessive hypertension may, as mentioned above, lead to dilatation.

The existence within the cerebral vascular tree of a tonic regulation depending on the blood pressure was suggested as early as 1900 by Biedl and Reiner (1900). Since then the subject had apparently not been studied until the last few years. Finesinger and Putnam (1933) concluded that the influence of the systemic arterial tension upon the cerebral vessels was insignificant, but the results mentioned above point to an opposite conclusion. The question must be considered as of importance in view of the modern theories of general circulatory regulation.

According to W. R. Hess (1930), who has given an excellent survey of the subject, the actual " goal " of circulation is the proper distribution of the volume of blood according to the immediate needs of each part of the organism. Under the term " nutritive reflex " he includes all the neurogenic, chemical, and physical reactions by which the blood supply of a definite organ increases when this is required by its immediate functional needs. Not only are changes in the vascular bed of the organ itself and in the cardiac output active at this time but other circulatory areas take part in the regulation through a " collateral vasoconstriction." This causes a reduction of the volume flow in the resting organs, enabling the blood to be diverted to the organs in activity. Parallel to this adjustment to local needs the general blood pressure level is maintained or even raised, as during muscular exercise, by a specific reflex mechanism, which Hess has termed " Entlastungsreflex." It is activated by the tonus of the sinus and aortic nerves. The auto-regulation of the blood pressure under control of these " buffer nerves " is not only active during exercise but also meets the requirements of gravity and of heat regulation.

If the cerebral vessels were subject to a tonic regulation through the " buffer nerves " they should take part in the mechanism of the " Entlastungsreflex." In other words, there should be a cerebral vasoconstriction when the blood pressure tends to fall during the activity of other organs of the body or when there was a change from the horizontal to the erect posture.

Cobb and Finesinger (1932) concluded from experiments on the vasodilator nerves of the pial arteries that stimulation of the central cut end of the vagus nerve produced pial dilatation through a direct reflex from the vagus to the facial nerve centre. Ask-Upmark (1935) arrived at similar results when the sinus nerves were stimulated. In consequence, the " buffer nerves " were thought by these authors to exert the same indirect action upon the pial arterioles as upon other vascular areas. On the other hand, previous investi-

gation (Fog, 1934, 1937, 1938*a*) has demonstrated that the response of the arteries on the surface of the brain to a fall or rise of blood pressure are identical whether all " buffer nerves " are cut or intact. Forbes, Nason, and Wortman (1937) found the vasomotor reactions to be in the nature of a dilatation when either the peripheral cut end of the vagus nerve or the sinus and aortic nerves were stimulated, an observation which indicated the " unspecific " nature of the response. Forbes, Nason, Cobb, and Wortman (1937) discovered that vasodilatation, following stimulation of the geniculate ganglion, was abolished after the local application of a solution of cocaine hydrochloride to the surface of the brain, whereas the reaction to a fall in blood pressure was preserved. In view of the last-mentioned results, it is justifiable to assume that the pial vessels do not take any part in a collateral vasoconstriction nor play an active part in the auto-regulation of the blood pressure, but that their tonus is decided by the level of the systemic blood pressure as controlled by the " buffer reflexes." This does not concern the possible action of metabolites produced during muscular or other exercise or the influence of a changing oxygen and carbon dioxide content of the blood.

The following experiments were planned to investigate the relationship between the blood pressure and the muscular tonus of the pial vessels more closely, and especially to study the influence of the activity of the sinus and aortic nerves in this respect.

Method

Cats, anæsthetised by butyl-bromallyl-malonylureid (Pernocton) were used. In all experiments artificial respiration was employed. The insertion of Forbes' cranial window and the measuring and recording of arterial diameters and of the blood pressure were accomplished as described in previous papers (Fog, 1934, 1937, 1938*a*). Variations of the blood pressure were produced by altering the blood volume. For this purpose a T-cannula was inserted into the abdominal aorta and connected to a special apparatus, shown in Fig. 1. A wide glass tube B (volume 200 c.c.), partly filled with defibrinated blood, connected above with a T-tube. One arm of the T-tube was connected to an oxygen tank C and the other to a vertical glass tube which dipped into mercury in the container A. This tube could be lowered and raised to any level under the surface of the mercury during the experiments, so the length of the tube submerged in the mercury (*a*) decided the pressure exerted upon the surface of the blood in the container B. Indirectly this pressure determined the systemic arterial tension of the experimental animal. When the tube in A was lowered blood would escape from B into the animal and the blood pressure be raised ; when the tube was raised a fall in systemic tension followed, as the blood ran from the animal into the apparatus.

In a number of cats the vagus, aortic, cervical sympathetic, and sinus nerves were divided on both sides. The total denervation of the carotid sinus was the most difficult part of the preparation. When the denervation was successful temporary compression of both common carotid arteries was not followed by a rise in general blood pressure ; if some branches of the sinus nerves were

left, arterial hypertension resulted. Cats prepared in this way and successfully denervated will henceforth be termed " denervated " animals.

In all 32 cats were experimented on. In eight animals successful section of the vagus, cervical sympathetic, aortic, and sinus nerves was carried out. Changes of the blood pressure level was carried out 105 times and the reactions of the pial arteries were studied. In some animals several rises or falls of identical extent were produced from various initial levels of systemic tension. In others, a gradual fall was brought about from the normal level to 60 or

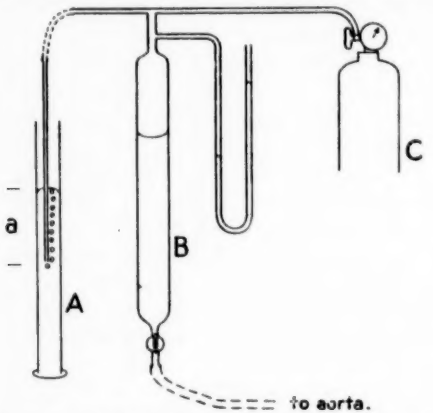

Fig. 1.—Apparatus for varying the blood pressure. A, mercury container. *a*, the height of mercury above the outlet for oxygen. B, blood container. C, oxygen tank. The apparatus was also used for compensating fluctuations of blood pressure in the experimental animal, see Fog (1938*b*).

70 mm. of mercury in the course of 30 minutes, whereupon the pressure was gradually raised to the original level or higher. Both types of experiments were carried out on " intact " as well as on " denervated " animals.

Results

The results are best illustrated by the study of some of the typical records. Fig. 2 represents the results in an " intact " animal. An arteriole under measurement presents a diameter of about 12–14μ with a normal blood pressure of 160 mm. Hg. During the gradual reduction of the circulating blood volume, the blood pressure falls to about 80 mm. Hg, and the diameter of the pial vessel becomes doubled. A subsequent rise of blood pressure to 160 mm. Hg brings the arteriole back to its original size. In Fig. 3 the results of a similar experiment performed on a denervated cat are presented and are seen to be essentially identical with those occurring in the " intact " animal (Fig. 2). In the denervated cat the rise of blood pressure, following the decrease, slightly exceeds the original level. In spite of this the artery did not contract further than to its original size. The responses of arteries larger than 100μ in diameter are best seen in another, intact, animal (Fig. 4). The variations of diameter are found to be in the same direction as in the smaller vessels,

but occur apparently more slowly. It will be noted that an elevation of tension above 150 mm. of mercury produces a narrowing of the lumen below its original diameter. Proportionally the vasomotor reactions in the larger vessels are far from being as pronounced as in the precapillary arterioles. In other animals the effect of sudden steplike rises of blood pressure from a low initial level

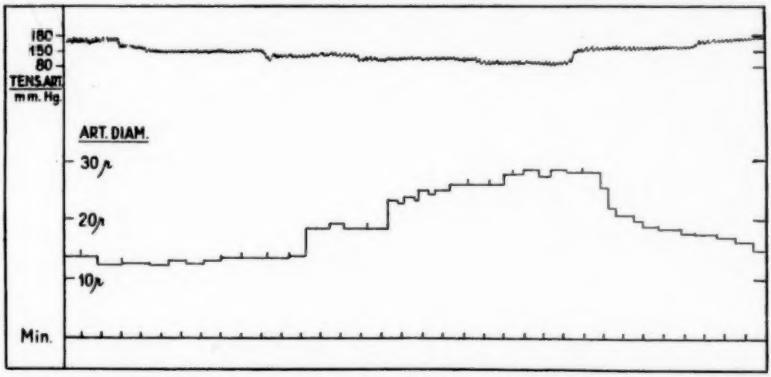

Fig. 2.—Reactions of a pial precapillary arteriole during decrease and increase of blood pressure, induced by variation of circulating blood volume. The vagi, sinus, aortic nerves, and the cervical sympathetic trunk intact.

was studied, of which Fig. 5 indicates the typical results. It is evident in this figure that the arteriole contracts most with the first rises of blood pressure.

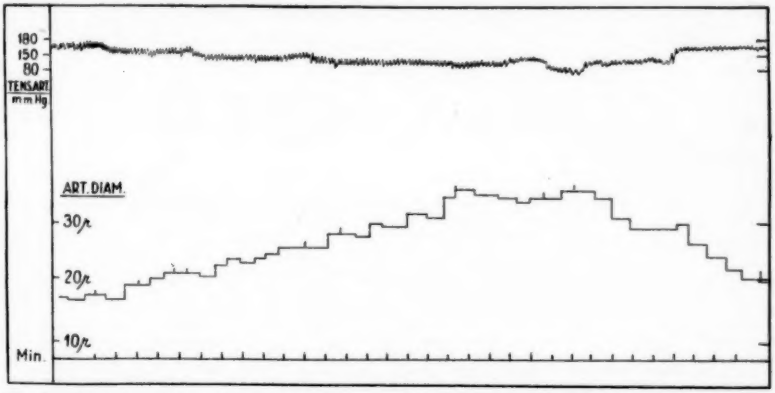

Fig. 3.—Reactions of a pial precapillary arteriole during decrease and increase of blood pressure. The vagi, sinus and aortic nerves, and the cervical sympathetic trunk severed.

These four records give the main features met with in the experiments. With variation of the intravascular pressure there was a change in diameter of the pial arteries. These results can only be interpreted as indicative of an active tonic regulation of the pial vessels. The tone is augmented when the blood pressure rises and is reduced when it falls. These reactions are inde-

pendent of the presence of the cervical sympathetic and the " buffer nerves." They are common to the precapillary and the larger arterioles, but are most pronounced in the former. As long as the systemic tension is about the normal level there are only slight variations of lumen ; an active alteration of tone must, however, be assumed, since there is no dilatation during rises or con-

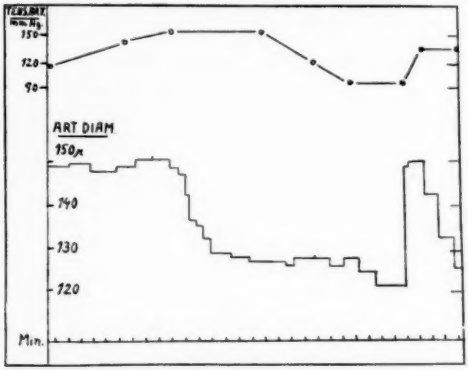

Fig. 4.—Reactions of a large pial artery during variation of blood pressure. " Intact " animal.

tractions during falls in pressure. The smaller arterioles are not reduced below their normal size even if the pressure is raised to 180 mm. of mercury, whereas the larger vessels in this case present some constriction. Obliterations were never seen. The level beneath which the tonic regulation entails obvious alteration of the diameter varies in different animals, but seems to be rather

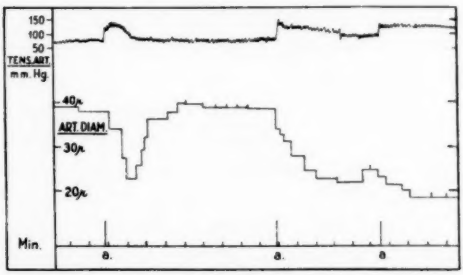

Fig. 5.—Reactions of a pial arteriole during sudden rises of blood pressure. " Intact " animal.

constant in any individual cat. Usually it was in the region of 110 mm. Hg, but occasionally as low as 90 mm. Hg.

Active responses were found to be pronounced in 24 cats. In another five animals the arterioles only presented slight alterations of diameter, not exceeding the error of the method of measurement ($\pm 2\mu$). As pointed out, this must be considered as a positive variation of vascular tone, which differs only quantitatively from that prevailing in cases where changes in diameter were

observed. The vessels of the remaining three animals, however, seemed to follow quite passively the fluctuations of the intravascular pressure, since the vessels distended during rises and narrowed during falls of arterial tension. According to the notes of the experiment one of these cats was under unusually deep anæsthesia. The two others had been exposed to great losses of blood during the preparation for the experiment and at the start of the experiments their arterial pressure was below 40 mm. Hg. It is to be mentioned that another three cats had just as low a blood pressure at the beginning of the experiment, and at first reacted passively, but after a normal level had been maintained by the pressure apparatus for some time, their vasomotor responses became active ; thus apparently the normal tonic regulation may be regained.

In several experiments it was determined how much the volume of blood in the container varied during the alterations of the level of the blood pressure. Thus a quantitative expression was obtained of the changes of blood volume which were necessary to produce a certain rise or fall of the systemic tension. Though this aspect of the problem will not be examined here, it may be mentioned that the amount was considerably less in the denervated animals, a finding which illustrates the " buffer " action of the aortic and sinus nerves. When the average number of cubic centimetres of blood led to or from the animals per 100 mm. of variations in blood pressure was computed from experiments on 18 cats it was found to be 100 to 115 c.c. in the intact as against 30 to 40 c.c. in the denervated cats. This only held for animals whose pial arteries were reacting actively. For the three cats with passive vasomotor responses the amount required was considerably larger, and in these cases it apparently made no difference whether denervation was performed or not.

Comment

The sensitiveness of the pial arteries to variations of the intravascular pressure is in the first place significant as an " emergency reaction," which comes into play when the auto-regulation of the systemic blood pressure fails, or, rather, is insufficient. It prevents, within certain limits, a rise of the capillary pressure during a rise in systemic blood pressure and thus protects the brain against injurious œdema. When the systemic blood pressure falls following hæmorrhage or shock this vascular reaction conduces to the maintenance of the cerebral blood flow by a reduction of the local peripheral vascular resistance.

Under various physiological conditions, moreover, these specific vasomotor responses are likely to be important factors, tending to keep the blood supply of the brain constant. The present series of experiments has confirmed the previous observation that the sinus and aortic nerves exert no influence upon the tonus of the pial arteries. Consequently, that vascular area will take no part in any of the functions which are elicited through the vasosensory nerves. This applies to the control of the blood pressure level, the adjustment of the circulation to local needs during exercise or other functional activity, and the effect of gravity during alterations of position. When, for example, the body

is moved from the horizontal to the erect posture, there is a slight rise of the blood pressure as measured at the height of the heart. At the same time a decrease of tension will occur within the cranial vessels on account of the hydrostatic factor. If this decrease is not fully compensated for by the general rise of pressure, the pial vascular tonus will be reduced and may so entail a diminution of the local arterial resistance. It is the actual intravascular tension in the cranium itself, whether it results from the general " buffer action " or from hydrostatic factors or from a joint action of both, which determines the tonic state of the pial vascular walls.

It has been suggested by Leonard Hill (1896) in his fundamental papers on the cerebral circulation that the cerebral supply is largely dependent upon the systemic blood pressure, as the neurogenic vasomotor control of this area is only weakly developed. This thesis is maintained in principle by several authors to-day. The demonstration of a moderate sympathetic innervation of the larger pial arteries and of specific vasodilatory nerves in the fifth cranial nerve does not speak against it, since the functional influences of these are not yet established. The absence of control by the aortic and sinus nerves also seems rather to support Hill's thesis. In my opinion, however, the responses of the pial arteries to variations of the endovascular pressure are important factors in an autonomous regulation of the cerebral circulation, as they will secure a relative independence of the cerebral blood-flow during fluctuations of the systemic pressure. In opposition to the point of view advanced by Hill and others one might suggest that the blood supply of the brain is less dependent on the systemic pressure than that of other organs which possess a strong sympathetic innervation and are largely controlled by the tonus of the vasosensory nerves. The last-mentioned organs take part in the maintenance of a rather constant level of general tension and their blood flow varies in the service of this function. The vascular resistance of the pial vessels, on the other hand, is regulated inversely when it is necessary to maintain the cerebral flow unaltered. Even a small change of diameter has a marked effect on the flow.

It must be borne in mind that the blood flow is determined by the relation between the arterial tension and the peripheral resistance. The amount of blood which passes through the pial vessels under varying conditions cannot be measured. Possibly the dilatation of a pial vessel following a fall of general pressure may not be sufficient to keep the flow at its original amount; again, if the blood pressure rises beyond the normal level the absence of augmentation of vascular resistance may not prevent an excessive blood flow. Thus the reactions of the pial vessels may act only to modify the influence of variations of systemic blood pressure, i.e. to establish a partial compensating mechanism. Finesinger and Putnam (1933), who measured the blood flow in the internal carotid artery and so determined the supply of a larger area than the brain surface, stated that the flow was closely related to the pressure in that artery. Similar results were arrived at by Bouckaert and Heymans (1933) and others.

It has been claimed by Heymans and Bayless (1937) that morphium-Pernocton-anæsthesia in dogs has a pronounced depressive action upon the

vasomotor reactions. In the present series all cats were anæsthetised by Pernocton, but the results did not differ from previous experiments in which chloralosane and other drugs were used (Fog, 1934, 1937). The responses dealt with in this paper are evidently not abolished by anæsthesia. Further, it was mentioned that there was a pronounced difference in the amount of blood required to provoke a definite variation of systemic pressure when the vaso-sensory nerves were intact and when they were cut. This indicates that the function of the aortic and sinus nerves is preserved in cats under pure Pernocton anæsthesia.

It is interesting to compare the " physical " tonic regulation with its chemical correlate, viz. the effect exerted on the cerebral vessels by a varying tension of oxygen and carbon dioxide in the blood (Wolff, Lennox, and Allen, 1930). It

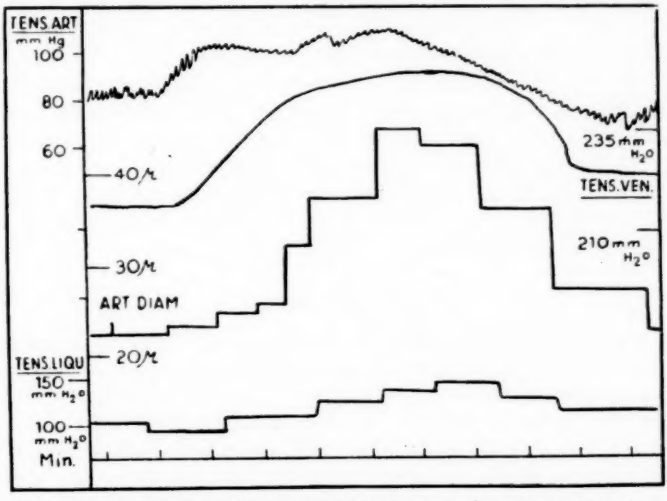

Fig. 6.—The reactions of cerebrospinal pressure, the diameter of a pial arteriole, the pressure of the external jugular vein and arterial pressure during asphyxia.

will suffice here to recall the pronounced dilatation of the pial arteries during asphyxia. In this condition there is a simultaneous rise of the systemic blood pressure ; thus the chemical and the physical regulation might be supposed to exert conflicting influences upon the cerebral vessels. It is evident from Fig. 6 that the action of the increased tension of carbon dioxide or the reduced oxygen tension or both together will be the predominant factors in such cases.

The nature of the tonic reactions which are elicited in the pial arteries and arterioles by variations of the intravascular pressure has not yet been established. If these reactions were only present with extreme changes of pressure, as when the blood pressure falls very low, they might be supposed to depend upon a coincident increase in carbon dioxide in the circulating blood, but as stated above the reactions come into play under physiological conditions, and further are present during normal respiration as well as during artificial ventila-

tion. An extra-cranial reflex mechanism is unlikely, in view of their independence of the cervical sympathetic and the sinus and aortic nerves. Forbes, Nason, Cobb, and Wortman (1937), as already mentioned, found the responses to a fall of systemic pressure preserved after a local application of cocaine to the pia, whereas vasodilatation following stimulation of the geniculate ganglion was abolished. In all probability, therefore, the sensitiveness depends upon some local mechanism. Forbes, Nason, and Wortman (1937) call attention to the fact that pial arteries smaller than 800μ have no vasa vasorum. Elimination of metabolites produced locally by the arterial muscle cells takes place by diffusion, and even a slight retardation of the flow through the vessels may involve an increase of carbon dioxide within the tissue of the arterial wall, which again may cause a dilatation. On the other hand, it has been demonstrated that an augmentation of tonus follows upon hypertension up to 180–200 mm. Hg ; in these cases significant variations of elimination are not likely to take place. The same authors recall the suggestion made by Bayliss (1902, 1923) that the smooth muscle of the arterial wall reacts to distension by a contraction, and *vice versa*. Bayliss in the year 1923 regarded the question as undecided, but the present observations seem to support his original point of view. In that case the responses must be interpreted as a general property of the arteries of the body. Recent experiments of Porsaa (1938) have shown that the arteries of the retina react in a similar way to fluctuations of internal pressure as do the pial vessels.

Summary

In a series of experiments on 32 cats changes of systemic blood pressure were produced 105 times by alteration of the circulating blood volume. The pial vessels contracted with a rise in systemic pressure and dilated with a fall. The pial vascular responses were identical whether the sinus and aortic nerves, the cervical sympathetic trunk and the vagi nerves were intact or severed.

The reactions of the pial arteries resulting from change of the endovascular pressure indicate the presence of a vasomotor regulatory mechanism within the limits of the cerebral vascular tree. This mechanism assists in keeping the blood flow to the brain constant during various physiological and pathological conditions of the general circulation.

The nature of the specific mechanism is discussed and it is suggested that it depends on properties inherent in the smooth muscles of the arterial wall.

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References

Ask-Upmark, C. E. F. (1935). " The Carotid Sinus and Cerebral Circulation." Copenhagen. *Acta Psychiat. Neurol.*, Suppl. 6.
Bayliss, W. M. (1902). *J. Physiol.*, **28**, 220.
—— (1923). *The Vaso-Motor System.* London.
Biedl, H., and Reiner, M. (1900). *Pflüg. Archiv. ges. Physiol.*, **69**, 158.
Bouckaert, J. J., and Heymans, C. (1933). *J. Physiol.*, **79**, 49.
Cobb, S., and Finesinger, J. (1932). *Arch. Neurol. Psychiat.*, **28**, 1,243.
Finesinger, J., and Putnam, T. J. (1933). *Arch. Neurol. Psychiat.*, **30**, 775.

Fog, M. (1934). Om Piarrteriernes vasomotoriske Reaktioner. Danish Dissertation, Copenhagen.
—— (1937). *Arch. Neurol. Psychiat.*, **37**, 351.
—— (1938*a*). *Ibid.*, in press.
—— (1938*b*). *Ibid.*, in press.
Forbes, H. S., Finley, K. H., and Nason, G. I. (1933). *Ibid.*, **30**, 957.
Forbes, H. S., Nason, G. I., Cobb, S., and Wortman, R. C. (1937). *Ibid.*, **37**, 776.
—— (1937). *Ibid.*, **37**, 334.
Forbes, H. S., and Wolff, H. G. (1928). *Ibid.*, **19**, 1,057.
Hess, W. R. (1930). *Die Regulierung des Kreislaufes*. Leipzig.
Heymans, C., and Bayless, F. (1937). *Arch. int. Pharmacodyn.*, **56**, 319.
Hill, L. (1896). *The Physiology and Pathology of the Cerebral Circulation*. London.
Porsaa, K. (1937). Unpublished data.
Wolff, H. G., and Lennox, W. G. (1930). *Arch. Neurol. Psychiat.*, **23**, 1,097.

SOME OBSERVATIONS ON THE DEPTH AND NERVE-CELL CONTENT OF THE SUPRAGRANULAR CORTEX IN NORMAL AND MENTALLY DEFECTIVE PERSONS

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Introduction

THE observations recorded in this paper have been made to test the validity of certain statements made by writers on mental deficiency as to the changes present in the cerebral cortex of the mental defective of no particular pathological type. While a small group of pathological rareties has received intensive study by neuropathologists, as regards the less distinctive cerebral abnormalities in the majority of institutional defectives the histologist is still in somewhat the same position as was the psychologist before the introduction of the Binet Scale of tests ; the frontiers of abnormality are as yet ill-defined. Nevertheless, in this latter group many microscopical abnormalities have been noted by various investigators, and to some of these findings reference may be made. According to Tredgold (1929), the nerve cells of the primary ament when compared with those of the normal cerebral cortex are characterized by: (1) numerical deficiency ; (2) irregular arrangement ; and (3) imperfect development of individual cells. These changes, he pointed out, are most obvious in the small and medium-sized pyramidal nerve cells. Bolton (1914), on the basis of micrometric measurements of three cortical areas in four ament and three normal brains, concluded that amentia is associated with " sub-evolution " of that part of the cerebral cortex which phylogenetically and ontogenetically is the last to develop, namely the supragranular layers, this sub-evolution being demonstrable by measurement of the cortical depth. Later observers have, however, failed to demonstrate such generalized reduced cortical depth in aments. In collaboration with Professor Berry (1934) I examined eight cortical areas in each of four defective brains from the point of view of depth of cell layers and numbers of nerve cells. The result of this investigation supported in the main Tredgold's views and not Bolton's. More recently Ashby and Stewart (1934) studied the depth of supra- and infra-

granular cortex in 62 defective and nine normal brains and were unable to demonstrate any statistically significant relationship between depth of cortex and mental ability.

During the last few years there has been accumulating at Stoke Park Colony a large collection of defective and normal brains. It has thus become possible to re-investigate some of the issues raised in the foregoing discussion, and observations have been made to provide, if possible, answers to the following questions : (1) Is thinness of the supragranular nerve cell layers a common feature of defective cortices ? (2) Is the average nerve-cell count in these layers reduced in defectives ? (3) Is the distribution of nerve cells throughout the section less uniform in defective than in normal brains examined by identical methods ? It is obvious that a statistical approach has advantages over an impressionistic one in evaluating the results of such an investigation.

Method of selecting Brains and Cortical Areas for Examination

Measurements of depth of supragranular cortex and counts of nerve-cell bodies in the supragranular cortex have been carried out in groups of some 30 ament and 30 normal brains. The ament brains were selected from the general collection at Stoke Park Colony in the following manner. All brains showing obvious macroscopic abnormalities, such as porencephaly, hydrocephalus, tuberose sclerosis ; lobar sclerosis and microgyria were excluded. Certain less obvious pathological types that had been encountered during routine microscopical examination, such as juvenile amaurotic idiocy and meningo-vascular and parenchymatous syphilis, were also excluded. According to this method of selection, therefore, Mongolian brains were included in the group to be examined. The majority of these ament brains came from defectives of the imbecile and idiot classes.

Three cortical areas from the left cerebral hemisphere have been examined in the normal and defective groups. They were from :

1. Bolton's " visuo-psychic " area (Area O.A. of von Economo). The part of this area which was examined lay immediately posterior to the point where the parieto-occipital fissure indents the convexity of the hemisphere.
2. The Frontal Pole (Area F.E. of von Economo). In this area the part selected for examination came from the convexity of the hemisphere at a point approximately half an inch equidistant from the great longitudinal fissure and the extremity of the frontal pole.
3. The Supramarginal Gyrus (Area P.F. of von Economo). The anterior and dorsal part of the gyrus was selected.

All these are examples of von Economo's six-layered type of isocortex. They all have a well-marked granular layer (L.IV) and the extent of the pyramidal cell layers of the supragranular cortex (L.II and L.III) may be clearly demarcated. It is in such association areas that Bolton and Tredgold have observed the most pronounced abnormalities which they have described as characteristic of the ament brain.

These areas have been taken from a total group of 39 normal and 54 defective brains. For reasons given later in this paper it was not found practicable to examine all three areas in every one of these individual brains. The smaller groups in which measurements and cell counts were made differed therefore among themselves as regards the constituent brains from which sections had been prepared. Since, however, the age distribution of these smaller groups did not materially differ from those of the main series given in Table I, this information has not been repeated in the subsequent detailed account of the findings in the three areas selected.

TABLE I

Age (yrs.) . .	0–4	5–9	10–14	15–19	20–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59	TOTAL
Normals . .	1	3	4	5	7	3	2	5	6	2	0	1	39
Defectives	1	5	14	14	9	5	3	3	0	0	0	0	54

Age distribution of total normal and defective groups.

Mean age of normals : 27 years. Mean age of defectives : 18 years.
Sex distribution : normals : ♂ 24, ♀ 15. Defectives : ♂ 27, ♀ 27.
Mental grade of defectives : feeble-minded 5, imbecile 30, idiots 19.

Technique

Method of preparing sections.—Both normal and defective brains were fixed in 10 per cent. formol-saline solution. In the case of the defectives, formol-saline solution was injected into the skull through the orbit as a routine procedure immediately after death. The time occurring between death and autopsy in the normal group was about 24 hours and in the defective group 24 to 48 hours. In removing blocks of tissue from the fixed brain the incisions dividing the gyrus were made as far as possible at right angles to the surface and also to the direction of the gyrus. The blocks of cortex so removed were stored in 5 per cent. formol-saline. Sections 25μ in thickness were cut on a freezing microtome and received into distilled water. The sections were then mounted on albuminized slides and allowed almost to dry in a warm atmosphere so that they adhered to the slide. They were then stained either with 1 per cent. aqueous Toluidin blue or 1 per cent. aqueous Cresyl violet, differentiated with Gothard's solution and absolute alcohol, cleared in xylol, and mounted in Mersol. In the event of a section becoming partially detached from the slide, this preparation was rejected and another stained. It was sometimes found that while satisfactory sections could be prepared in this way from one area, the sections cut from another area of the same brain were unsatisfactory, either because of œdema or poor staining of the nerve cells or because of the appearance of small holes in the glial ground substance. Such artefacts, which were encountered more frequently in the defective group, made accurate cell counting impossible. All sections exhibiting these appearances were therefore rejected and replaced by others from brains that proved

to be satisfactory. It thus came about that the constitution of the groups of normal and defective brains varied somewhat according to the cortical area examined.

Method of measuring depth of supragranular cortex.—Measurements were confined to the crowns of the gyri. The area of the section in which measurements were to be taken was determined in the following manner. The lines of demarcation between cortex and central core of white matter on either side of the section, corresponding to the walls of the gyrus, were marked on the back of the slide with a blue pencil. These two lines were then extended so as to mark off an area in the crown of the section. The slide was then transferred to the movable stage of the microscope (Leitz objective No. 3, draw tube at 170 mm.), and a number of measurements taken of the combined depths of layers II and III in the previously demarcated area of the cortex, a Leitz " Schraubenmikrometer " being used (eyepiece $\times 10$). The left-hand blue line having been recognized, a measurement was taken in a vertical direction between the most superficial point of the external granular layer (L.II) and the base of the deepest large pyramidal cell of L.III. The slide was then moved slightly to the left and another measurement made, this time working from below upwards. In this way a number of estimations of the combined depth of these two layers of the cortex were made until the right-hand blue-pencil line came into view. The extent of the demarcated area of the crown of the gyrus necessarily varied between sections, so that a varying number of these measurements (on the average 15) were made per section. The individual measurements were recorded in units of the scale of the micrometer eyepiece. At the magnification used one division of the scale=41·66μ.

Method of cyton counting.—After preliminary trials on small groups of normal and defective sections, it was decided to adopt the following method. The cyton counts were confined to the supragranular cortex and to the middle of the pyramidal nerve cell layers. The depth of L.II and L.III was first estimated as described above and the midpoint of this depth of cortex brought into the centre of the microscopical field. The micrometer eyepiece was then exchanged for a Leitz Ehrlich ocular $\times 6$ set at position 6. The objective of the microscope was then changed for a No. 6 and the number of nerve-cell bodies visible in the square field counted. The side of this square measured 200μ. All neuronic cytons, whether large or abnormally small, were counted. In some of the defective sections difficulty was occasionally experienced in deciding whether a cyton belonged to a minute neurone with little stainable cytoplasm or to a glial cell. In such cases the shape and character of the nucleus was taken as distinguishing features, a nucleus having a nucleolus being considered as identifying a neuronic cyton. Seven such cell counts were made in the crown of the section. Owing to the method adopted it was impossible to tell beforehand whether one was going to count in an area of the cortex possessing numerous or few nerve cells. Any bias in the selection of microscopic fields was therefore avoided. If the area contained a large blood vessel a count was not made, and another position found. This rule was adopted in both normal and defective sections.

In examining the parietal area (P.F.), in addition to the method of cyton counting described above, a more extensive survey of the crown of the section was made. Instead of seven counts using a large square, 20 counts per section were made using a smaller square in the eyepiece. The sides of this square microscopic field measured 100μ. Apart from this difference, precisely the same method of counting in the middle part of L.II and L.III was adopted.

Measurement of Depth of Layers II and III in Normal and Defective Cortices

In none of the three selected areas has measurement of the supragranular cell layers demonstrated a significant difference in mean between normals and defectives.

TABLE II

	AREA O.A.	AREA F.E.	AREA P.F.
No. of normal sections	29	27	32
No. of defective sections	34	27	32
Normal mean	22·50	23·78	26·51
Defective mean	21·09	23·64	25·94
Difference in mean	1·41	0·14	0·57
Standard error of difference ..	±0·74	±0·99	±0·93
Difference÷S.E.	1·90	0·14	0·61
Significance	—	—	—

Comparison of mean depth of cortex.

It will be seen in Table II that only in the visuo-psychic area O.A. was a difference approaching significance discovered. While it is possible that with much larger numbers of cases a result in favour of the normals would have been obtained, such small differences would have little meaning from the neurological point of view. In any case, such differences may well be regarded as the expression of the fact that normal brains are on the average considerably larger than those of defectives. Thus the hypothesis that amentia is associated with a specific reduction in depth of the supragranular cortex receives no confirmation from these findings.

Cyton Counts in the Middle of Layers II–III in Normal and Defective Cortices

Comparison of mean cyton counts.—In contrast to the measurements of cortical depth, the method of cell counting revealed an important difference between the two groups of brains.

TABLE III

	AREA O.A.	AREA F.E.	AREA P.F.
No. of normal sections	30	27	30
No. of defective sections	38	27	32
Normal mean	21·19	16·71	16·62
Defective mean	25·23	18·68	15·08
Difference in mean	4·04	1·97	1·54
Standard error of difference ..	±1·23	±1·16	±1·01
Difference÷S.E.	3·28	1·70	1·52
Significance	++	−	−

Comparison of mean cyton counts.

It will be seen in Table III—area O.A.—that the number of neuronic cytons per unit area in the defective sections significantly exceeded the normal figure. To avoid any possible misconception as to the meaning of this observation it may be emphasized that the nerve cells counted in the defective sections included many examples of small cell types, that this crowding together of defective cytons is to be regarded as an indication of cerebral inferiority and is in marked contrast to the well-developed, well-spaced-out cells of the normal cortex.

Variability of cyton counts.—The most striking and constant differences between the two groups of brains were found when the results of the cell counts were analysed for differences in variance (i.e. a measure of variability, the mean square deviation from the mean or standard deviation squared). The total variance may be split into two parts : the " variance within sections," which gives a measure of the amount by which the separate cell counts made in the same section vary among themselves ; and the " variance between sections," which gives a measure of the amount by which the average cell counts of sections vary between themselves from section to section, i.e. in this series from brain to brain. The former variance thus indicates whether the nerve cells are distributed with more or less uniformity throughout the part of the gyrus sampled by the cell counts. The variances of the cell counts, having been calculated in the normal and defective series, were then compared using Fisher's z method (1934). The term z is derived from the difference between half the Naperian logarithm of each variance. The significance of the difference between the variances was then ascertained either by using the table of z or, when the number of observations was large and not very unequal, by calculating the standard error of z.

Tables IV, V, and VI show that the variance between sections was significantly greater in all three areas among the defectives than among normals. This may be taken as an indication that certain cortical areas from a series of defective brains such as those examined in this investigation show a greater variability in the average nerve-cell content of the supragranular cortex as compared with normal brains. While it is true that on the average there were more cytons per unit area in the defective group, the defective series differed from the normal not only in providing sections in which the nerve cells were more crowded together than in any normal, but also in showing examples of greater sparseness of nerve cells than did any normal.

TABLE IV

		DEGREES OF FREEDOM	SUMS OF SQUARES	MEAN SQUARE	$\frac{1}{2}\log_e$
Normals	Within sections	180	2,544·00	14·13	1·3242
	Between sections	29	1,801·76	62·13	2·0647
Defectives	Within sections	228	5,495·17	24·10	1·5911
	Between sections	37	12,036·4	325·3	2·8924

Analysis of variance. (Area O.A.)

	z	S.E.	z/S.E.	SIGNIFICANCE
Within sections	0·2669	0·07	3·81	++
Between sections	0·8277	0·18	4·60	++

Comparison of variance of normals and defectives.

TABLE V

		DEGREES OF FREEDOM	SUMS OF SQUARES	MEAN SQUARE	$\frac{1}{2}\log_e$
Normals	Within sections	162	1,519·99	9·383	1·1195
	Between sections	26	1,923·04	73·96	2·1518
Defectives	Within sections	162	2,403·42	14·84	1·3487
	Between sections	26	4,635·75	178·3	2·5918

Analysis of variance. (Area F.E.)

	z	S.E.	z/S.E.	SIGNIFICANCE
Within sections	0·2292	0·079	2·90	++
Between sections	0·4400	0·196	2·24	+

Comparison of variance of normals and defectives.

TABLE VI

		DEGREES OF FREEDOM	SUMS OF SQUARES	MEAN SQUARE	$\frac{1}{2}\log_e$
Normals	Within sections	180	2,610·53	14·50	1·3371
	Between sections	29	1,914·58	66·02	2·0949
Defectives	Within sections	192	3,007·99	15·67	1·3759
	Between sections	31	4,846·56	156·3	2·5259

Analysis of variance. (Area P.F.)

	z	S.E.	z/S.E.	SIGNIFICANCE
Within sections	0·0388	0·073	0·53	–
Between sections	0·4310	0·183	2·36	+

Comparison of variance of normals and defectives.

The most important finding, however, was that in two areas the variance within sections was significantly greater in the defective group, while in the third area the difference though non-significant was in the same direction. This means that within a single section the cytons tended to be more irregularly grouped in the defectives than in the normals. This abnormal and irregular arrangement of nerve cells is often easily distinguishable in defective sections by ordinary microscopical inspection and has been reported by other observers. To reduce this qualitative observation to terms of measurement is not an easy task, and while it is probable that many different methods carried out upon sufficient numbers would yield a significant difference between defectives and normals, the actual magnitude of the difference found would depend upon how accurately such methods estimated the greater irregularities of the defective group. This point is well illustrated in the following re-examination of area P.F., using in this case a counting square of smaller dimensions and counting more fields (see Technique).

It will be seen in Table VII that the new method of counting has yielded highly significant differences between the two groups as regards variances between and within sections. The magnitude of such observed differences thus depends largely on the refinement of the technique employed, and no doubt further research would improve the arbitrary methods employed in this investigation—which have, nevertheless, sufficed to detect significant differences between the two groups of brains.

A comparison of the results of cyton counts in Mongolian, non-Mongolian defective and normal sections.—The foregoing analyses have demonstrated differences between normal cortices and those of a mixed group of defectives including Mongols. It was considered profitable to analyse the results of the cell counts in area O.A. for the three groups separately in order to discover

to what extent the abnormalities of the defective group were due to the admixture with Mongols. The results of this comparison are given in Table VIII.

TABLE VII

Area P.F. (Smaller counting square.)

Comparison of mean cyton counts: normals 3·8891; defectives 3·9266. Difference 0·0375 ± 0·2112 (0·18 × S.E.) non-significant.

		DEGREES OF FREEDOM	SUMS OF SQUARES	MEAN SQUARE	½ log
Normals	Within sections	608	1,697·25	2·792	0·5134
	Between sections	31	179·87	5·802	0·8791
Defectives	Within sections	608	2,441·25	4·015	0·6950
	Between sections	31	704·29	22·72	1·5617

Analysis of variance. (Area P.F.)

	z	S.E.	z/S.E.	SIGNIFICANCE
Within sections	0·1816	0·041	4·43	++
Between sections	0·6826	0·180	3·79	++

Comparison of variance of normals and defectives.

TABLE VIII

	NORMAL	MONGOL	NON-MONGOLIAN DEFECTIVES
No. of sections	30	11	27
Mean	21.19	24·45	25·55
Difference in mean		3·26	1·10
S.E. of difference		±0·76	±1·51
Difference ÷ S.E.		4·29	0·73
Significance		++	−

Comparison of mean cyton counts in normal, Mongolian and non-Mongolian defective cortices. (Area O.A.)

The results of this analysis indicate that the number of cytons per unit area in the Mongol sub-group significantly exceeded the normal figure but did not differ significantly from that of the non-Mongolian defective group. Thus the relative crowding together of nerve cells was a feature common to both groups of defectives. The analysis of variance of cell counts in the three groups is shown in Table IX.

TABLE IX

		DEGREES OF FREEDOM	SUMS OF SQUARES	MEAN SQUARE	$\frac{1}{2}\log_e$
Normals	Within sections	180	2,544·00	14·13	1·3242
	Between sections	29	1,801·76	62·13	2·0647
Non-Mongolian defectives Mongols	Within sections	66	1,463·45	22·17	1·5494
	Between sections	10	2,155·66	215·6	2·6868
	Within sections	162	4,031·73	24·89	1·6073
	Between sections	26	9,815·05	377·5	2·9668

Analysis of variance. (Area O.A.)

	z	1 PER CENT. POINT	5 PER CENT. POINT	SIGNIFICANCE
Within sections	0·2252	0·226	0·161	++
Between sections	0·6221	0·538	—	++

Comparison of variance of normals and Mongols.

	z	1 PER CENT. POINT	5 PER CENT. POINT	SIGNIFICANCE
Within sections	0·0579	—	0·178	—
Between sections	0·2800	—	0·501	—

Comparison of variance of Mongols and non-Mongolian defectives.

It will be seen that, as in the total defective group, the variance of counts within and between sections was significantly greater in the Mongol group than in the normal. When, however, the Mongol and non-Mongol defective groups were compared, no significant differences in variability of counts either within or between sections was discovered. The greater variability of the total defective group compared with the normal cannot, therefore, be attributed to the inclusion of Mongols.

Discussion

This investigation has shown that in a representative group of institutional defectives of no particular pathological type abnormalities of cortical structure may be demonstrated by a relatively simple and arbitrary technique. The main negative finding was that micrometric measurements of the depth of the supragranular cortex failed to reveal any appreciable difference between normal and defective sections. Bolton's conclusion, that as a general rule this part of the cortex is thinner in mental defectives, was not, therefore, con-

firmed. The results of the neuronic cyton counts, on the other hand, indicated that, in the main, two processes making for abnormality had been operative in the defective brains : the one producing an excessive crowding together of the nerve-cell bodies, the other producing the opposite effect, an undue sparseness leading to a low cell count. These effects were to be seen not only in the average cyton content (variability between sections), but also in the patchy distribution of cytons within the section itself (variability within the sections).

Before discussing the neurological significance of these abnormalities it may be pointed out that in the development of the normal cerebral cortex several factors governing the number and arrangement of the constituent neurones come into play. As is well known, the development of the crowded fœtal cortex into the adult state, where the nerve cells are well spaced out, is associated primarily with the rapid surface expansion of the growing cerebral hemispheres (Donaldson, 1895). Besides this general and largely mechanical process, there are inherent factors which produce the final organization of the numerous cortical areas appropriate to the species. In man von Economo and Koskinas (1925) have distinguished 52 principal cortical areas, each with a characteristic structure which may be defined in terms of depth of layers and size and number of the neuronic cytons composing these layers. There are also other, less easily measured, differences between these areas. For example, in the deeper part of L.III in area O.A. the nerve-cell bodies are normally arranged in short columns, while in area F.E. this columnar distribution is much less evident. That the appearances of anatomically identical areas from presumably normal brains may vary considerably in ordinary post-mortem material is evident from the results of the present investigation, but it is noteworthy that in defective brains this variability is considerably greater. The conclusion may legitimately be drawn that some at any rate of the differences in mentality existing between normal and mentally defective persons are due to the greater frequency and degree of such structural abnormalities in the defective group.

As has already been stated, the increased variability of the cyton counts in defectives may be attributed to the operation of two processes, one making for sparseness, the other for crowding together of the nerve-cell bodies. Taking the group as a whole, the latter factor appears to be the more common and may be interpreted as a sign of inhibited development of the ament brain. This relative crowding together of cytons may thus be regarded as the microscopical counterpart of the small size of the majority of these defective brains as compared with the normal. In this respect, then, the defective brains show signs of retarded neurone development.

With regard to situations in the defective brains in which a paucity of nerve cells per microscopic field was found, it may be said that the problem of pathogenesis is much more complex. There is no analogy here with the fœtal state. The wider separation from each other of the normal adult nerve-cell bodies is due in part to the greater development of their processes, the cell bodies and nuclei being larger as a general rule in pyramidal nerve cells the processes of which traverse a longer course in the nervous system (Bok, 1936).

It was certainly not the case that the widely separated cytons in those sections from defectives which exhibited nerve-cell paucity were better developed than those present in more normally populated areas. Thus the low cyton counts per microscopic field cannot be attributed to a compensatory hyper-development of the nerve cells. Areas of relative nerve-cell poverty were not uncommonly encountered in some of the normal brains, and as is well known the middle of the third layer of the cortex seems to be peculiarly liable to exhibit such changes. The patchy " areas of clearing " in the defective cortex cannot, therefore, be considered to be a change peculiar to mental defectives, but merely occur more frequently and often to a greater degree than in the normal series. It is probable that many ætiological factors are operative in setting up this picture of relative nerve-cell deficiency. Some of the less conspicuous areas devoid of nerve cells may have resulted from a malformation of the cortical pattern : that is to say, in the final stage of organization less than the appropriate number of nerve cells may have developed in that part of the cortex. It will be recalled that in the middle and inferior temporal convolutions of the normal brain the nerve cells in L.III are arranged in a patchy way that would constitute an abnormality if found in other parts of the cortex. Thus microscopic fields poorer in nerve cells than fields in the immediate vicinity may arise in a gyrus without the intervention of destructive influences. Some of the patchy " areas of clearing " in the defective cortices may thus result from faulty development. On the other hand, in several instances the poverty of nerve cells was too gross and generalized to admit of this explanation, quite extensive fields having the appearance of cortical devastation being sometimes observed in the present series of defective brains ; and this even though the method of selecting the brains was calculated to exclude cases where such changes might have been expected. It is by no means clear what significance is to be attached to such presumably degenerative phenomena, since it is difficult to assess their chronological onset. If such lesions occur during the developmental stages of the cortex they may well play a primary part in the complex ætiology of amentia ; if, on the other hand, they are merely coincidental and appear in later life as the expression of the less effective resistance on the part of a poorly developed organ to environmental stress, their ætiological significance is of secondary importance. Only a detailed examination of individual cases can be expected to throw light on this problem.

Finally, it may be pointed out that while the micrometric measurements of cortical depth recorded in this paper have not confirmed Bolton's views as to the anatomical basis of amentia, the abnormalities which have been demonstrated in these defective brains lend support to the spirit if not to the letter of his thesis : namely, that mental deficiency is commonly associated with structural anomalies in those parts of the nervous system which phylogenetically and ontogenetically are of recent acquisition.

Summary

A microscopic examination of sections from three cortical areas (O.A., F.E., and P.F. of von Economo) has been carried out in groups of some 30

normal and 30 mentally defective brains. Brains exhibiting gross pathological macroscopic appearances were excluded. The combined depth of layers II and III of the supragranular cortex has been measured and a series of neuronic cyton counts made in the middle of this pyramidal cell lamina.

No significant difference in mean depth of cortex was demonstrated between normals and defectives.

There was no indication that a general poverty of nerve cells was an important characteristic of this group of ament brains. On the contrary, in the visuopsychic area the average number of cytons per microscopic field in the series from defectives significantly exceeded the normal figure. This diminution of normal spacing between poorly developed nerve cells is highly characteristic of the defective cortex. Cellular richness *per se* is not, therefore, in this case to be equated with functional superiority.

Microscopic fields poor in nerve cells were more often found in ament than in normal cortices, but the general effect of such cases was not sufficiently marked in the present investigation to reduce the average nerve-cell count below the normal level.

The distribution of neuronic cytons throughout the middle portion of the supragranular cortex in defectives was significantly less uniform than in the normals. This irregular arrangement of nerve cells in the mental defective, suspected from the qualitative observations of earlier observers, finds confirmation in the results here presented.

The average nerve-cell content of sections was significantly more variable in the defective group than in the normal.

No significant differences were found between Mongol and non-Mongolian defectives as regards mean cell content or variability of nerve-cell distribution

This investigation supports the view that amentia is commonly associated with structural abnormalities of those parts of the neopallium which have been most recently acquired.

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REFERENCES

Berry, R. J. A., and Norman, R. M. (1934). *J. Neurol. Psychopath.*, **14**, 289.

Bok, S. T. (1936). *Verhandelingen der Koninklijke Akademie van Weltenschappen te Amsterdam Afdeeling Natuurkunde* (tweede sectie), Deel XXXV, No. 2.

Bolton, J. S. (1914). *The Brain in Health and Disease*, Chapter 6. London : Edward Arnold.

Donaldson, H. H. (1895). *The Growth of the Brain*, p. 238. London : Walter Scott.

Economo, C. von, and Koskinas, G. N. (1925). *Die Cytoarchitektonik der Hirnrinde des erwachsenen Menschen*. Wien u. Berlin : Julius Springer.

Fisher, R. A. (1934). *Statistical Methods for Research Workers*. 5th ed. Edinburgh : Oliver and Boyd.

Stewart, R. M. (1934). *Proc. roy. Soc. Med.*, 28, **1**, 786.

Tredgold, A. F. (1929). *Mental Deficiency*. 5th ed., p. 78. London : Baillière, Tindall and Cox.

THE RELATIONSHIP IN MAN OF CEREBRAL ACTIVITY TO BLOOD FLOW AND TO BLOOD CONSTITUENTS

BY

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THIS title limits us to a discussion of the human subject. In addition we shall not deal with pathological states of either the brain or the cerebral blood vessels, nor shall we stop to point out how little is known about the subject of vascular morphology in relation to intelligence. As a preliminary to our paper we wish to clarify a prevailing confusion of thought regarding the relative importance of the central nervous and the cardiovascular systems. Too many look on the brain as entirely at the mercy of the circulation and helpless in the face of failing blood-borne supplies. The relationship between brain and blood is reciprocal, but emphatically is one in which the brain is master ; the circulation is servant.

We shall briefly summarize certain findings which bear on the title of this paper.

I. Cerebral Activity and Total Blood Flow

If this subject were being presented in the last century, physiologists and clinicians alike would have stressed the importance of circulation. Our medical ancestors had the belief that humours were important determinants of behaviour, and our immediate predecessors interpreted many nervous and mental states in terms of cerebral blood flow. Plethora and cerebral congestion was the cause of multiple symptoms calling for blood-letting. On the other hand, cerebral anæmia was believed to cause phenomena ranging from sleep to fits. It is now realized that changes in the normal activity of the brain, even to the point of abnormality, ordinarily are the cause and not the result of a concomitant alteration in cerebral blood flow.

Activity of muscle is accompanied or followed by the opening up of the capillary bed in the muscle. Similarly, it might be expected that increased mental activity is accompanied by an increased cerebral flow. In this important

field, observations are all too scanty. While subjects were doing sums in mental arithmetic, Lennox and Leonhardt (1931*a*) measured the oxygen content of blood leaving the brain by the way of the internal jugular vein, and Carmichael, Williams, and Doupe (1937) recorded the alterations in cerebral pressure by means of a needle in the cistern. Both of these methods, though open to objections, pointed to an increase of blood flow during mental work, the result probably of an increase of systemic blood pressure. As for localized changes, Fulton (1928) noted an increase in the bruit when a patient with an arterio-venous aneurysm of the occipital lobe read in a dim light. In all cases, however, these circulatory increases are the result of cerebral activity, and not the cause. Such changes in total flow as occur are less than those which are constantly occurring fortuitously, and they are probably not significant. Also, if blood pressure and flow are artificially but only moderately decreased, mental performance is not affected. Finally, drugs which greatly alter central nervous system activity, notably the sedative, do not, according to the flow recorder experiments of Lennox *et al.* (1935*b*) and of Gibbs *et al.* (1935*a*), significantly alter cerebral blood flow unless there is coincident change in systemic blood pressure.

Particularly interesting are those manifestations which occur intermittently and, usually, involuntarily. Are they the result of a sudden decrease in cerebral flow ? In observations by means of a thermo-electric flow recorder inserted in the internal jugular vein it was found that no alteration in flow took place with the onset of natural sleep or with attacks of narcolepsy (Gibbs *et al.*, 1935*b*), or with seizures either of the petit mal or the grand mal type (Gibbs *et al.*, 1934). In the case of grand mal, blood flow was greatly increased during the seizure, due to the increase in blood pressure and the accumulation of carbon dioxide in the blood. Also, the oxygen content of the blood leaving the brain was not sufficiently low to be directly responsible for the symptoms (Lennox *et al.*, 1936*a*). These observations deal with alterations in the total blood flow of the brain. There may be spasm of vessels in localized regions which would not affect the total flow, but would be of clinical significance. As regards epilepsy, there is weighty evidence against this hypothesis of localized vascular spasm.

II. Effect of Temporary or Functional Alterations of Flow

In this and the succeeding section we shall deal with evidences of two kinds pertaining to cerebral activity : first, consciousness, and second, the electrical activity of the cortex.

More attention is commonly paid to a decrease than to an increase of blood flow in the head. Consideration of the subject is complicated because the symptoms are different in acute and in chronic anæmia. Apparently in states of blood deficiencies which are gradual in onset, the body or the brain (or both) make adjustments which, in spite of the handicap, permit brain function to continue. The mechanism of the adaptation is a subject which requires future study. Defective circulation to the brain with resultant decrease in cerebral

activity can be brought about either by decreasing the arterial pressure or by increasing the venous pressure. We shall first discuss observations in which blood pressure was drastically reduced.

In the upright position, the cerebral circulation is maintained only by active neuro-vascular adjustments which are easily deranged. We have recorded both blood pressure and cerebral (internal jugular) blood flow in patients with orthostatic hypotension (natural or induced) and with irritable carotid sinus reflex. In these patients, unconsciousness was preceded by great fluctuations in cerebral blood flow and blood pressure, and was accompanied by a plummet-like drop in both (Lennox, *et al.*, 1935*a*).

Of particular interest are observations of the cortical electrical activity of subjects when the cerebral blood flow is acutely reduced. We have recently

CHANGES IN THE ELECTRO-ENCEPHALOGRAM WITH SYNCOPE
FROM CRITICAL FALL IN BLOOD PRESSURE (CAROTID SINUS REFLEX)

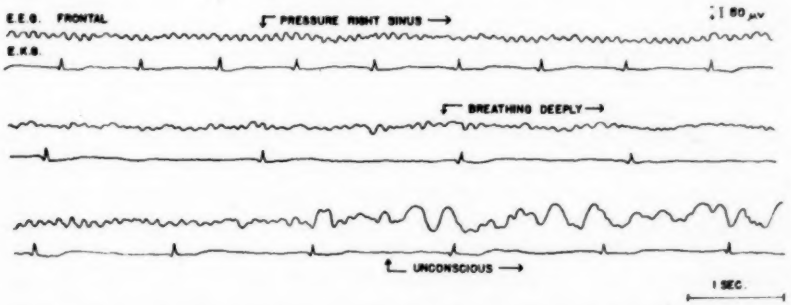

Fig. 1.—Changes in the electro-encephalogram with syncope due to a critical fall in blood pressure produced by pressure on a hyperactive carotid sinus reflex. Record labelled *E. E. G.* is the electro-encephalogram from the mid-frontal region (indifferent lead on the ear). The simultaneous record of the heart rate is marked *E. K. G.* The tracings are continuous in time ; one-second time calibration is at the lower right corner. The excursion of the electro-encephalograph with 50μv is given at the upper right corner. Signals as labelled on the record.

amplified the preliminary observations of Gibbs, Davis, and Lennox (1935) by taking simultaneous electrocardiograms and electro-encephalograms. The results are much the same in cases of orthostatic hypotension and of carotid sinus reflex of the cardiac type. The cardiac standstill and the acute fall in blood pressure in the carotid sinus cases are not at first followed by changes of the electrical rhythm of the brain, other than a somewhat faster rhythm which accompanies states of increased attention. Six or eight seconds after the heart has stopped beating consciousness is lost abruptly, and at the same time the rate of electrical waves is reduced from the normal eight to twelve a second to an abnormal three to four a second. Fig. 1 illustrates a case in which, following pressure over the sinus, there was bradycardia and a fall of 50 mm. Hg. in the systolic blood pressure. Consciousness was lost after 16 seconds, simultaneous with a great slowing of the rate of cortical electrical

waves. What is not shown in this figure is that shortly after the appearance of the large slow waves, which become progressively slower, the record flattens ; the large slow waves reappear, however, and increase in frequency as the subject returns toward consciousness, eventually to regain the normal frequency and amplitude as full consciousness is regained.

As for the effect of an increase of cerebral venous pressure, the extent to which the observed mental changes associated are due to inadequate supply of blood and how much to the direct injury of nerve cells has been inadequately studied. Patients in coma from a cerebral injury or with a tumour do not necessarily have an abnormally low oxygen saturation of the blood in the internal jugular veins (Lennox and Williams, 1938). This does not prove, however, that the brain as a whole has had an adequate supply of blood and oxygen. The blood supply to certain areas may have been cut off, leaving a normal blood flow through other areas. However, although the circulatory disturbance may not be widespread, the alteration in the activity of nerves, as indicated both by neurological tests and by a study of cerebral action potentials certainly is widespread. The important question, therefore, would seem to be, not is there a local disturbance of a circulatory nature, but, rather, how does such a local disturbance interfere with activity throughout the whole brain.

III. The Importance of Various Constituents of the Blood

Blood is necessary to the brain not because of its mass but because of what it contains. An important but inadequately explored field is the effect on function of the brain of an increase or a decrease of a single constituent of the blood. The metabolism of the brain differs in certain features from the metabolism of other tissues, and the nerve cells are unusually sensitive to alterations of their food supplies. The electrical activity and hence the proper functioning of the many neurones is primarily dependent upon the tensions of oxygen and of carbon dioxide, the concentration of glucose and of electrolytes, and the permeability of their membranes. Likewise Dusser de Barenne *et al.* (1937) have demonstrated the close correlation between the *p*H of the cortex and its electrical activity. Observations in human subjects have been limited ; inferences must be drawn with caution because the particular constituent or event being measured is but one link of a closely joined and mutually interacting chain of events. Observations which we shall now report are but preliminary.

Carbon dioxide.—In affairs of the brain, carbon dioxide has received less publicity than oxygen. Yet if we confine ourselves to states of cerebral activity in which consciousness is present, we find that variations of carbon dioxide produce greater changes in the rate both of cerebral blood flow and of cortical electrical waves than variations in oxygen. Lennox and E. L. Gibbs (1932) observed in human subjects that an increase in the carbon dioxide content of arterial blood caused a great and invariable increase in blood flow through the brain and coincidentally a decrease in the flow of the leg. Decreasing the carbon dioxide content in arterial blood by voluntary hyperpnea caused the opposite effect (Fig. 2). The findings are supported by changes in the colour

of retinal veins observed by Cobb and Fremont-Smith (1931). In sharp contrast, we observed that changes in the oxygen content of arterial blood resulted in moderate changes in cerebral blood flow, usually without reciprocal alterations in brain and leg. These findings, based on the gaseous content of arterial and venous blood, were later substantiated by Gibbs *et al.* (1935*c*) by means of the thermo-electric flow recorder. The beneficial effect of an increased concentration of carbon dioxide is remarkable. Mental performance, impaired by anoxæmia, is restored by the addition of 3 per cent. carbon dioxide

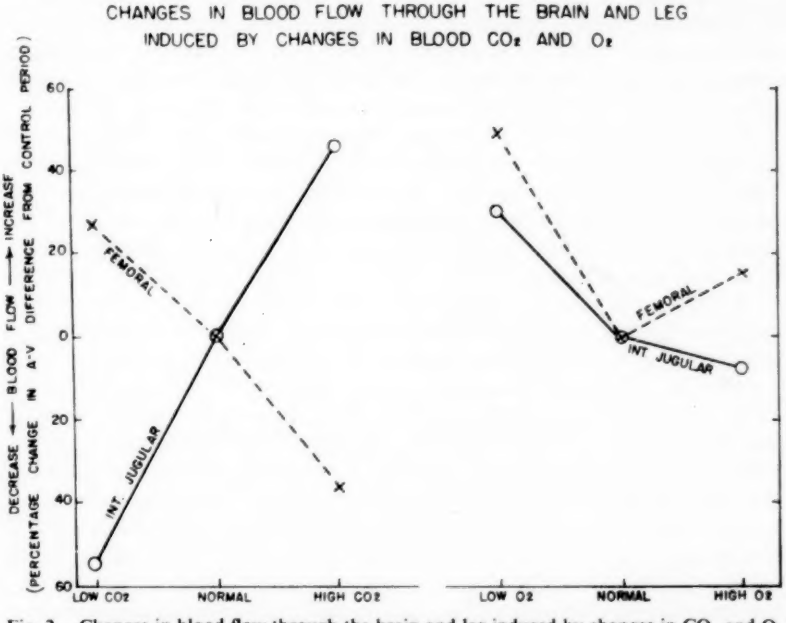

Fig. 2.—Changes in blood flow through the brain and leg induced by changes in CO_2 and O_2 content of arterial blood. The values are the average of from four to ten experiments in which subjects breathed high or low concentrations of either CO_2 or O_2 (four procedures). Blood was drawn from an artery or an internal jugular vein before and during the procedure, and the A-V difference calculated. In both figures the circle at the centre marks the preliminary values, and the end of the line the values during the breathing. The ordinate measures the percentage change in the A-V difference from the values obtaining in the preliminary period. Positive percentages indicate a decrease in A-V differences and an increase in the speed of blood flow. Solid lines indicate changes of flow in an internal jugular vein, dashed lines changes in a femoral vein.

to the air breathed (Gellhorn, 1937). Patients can be roused from simple stupor (Loevenhart *et al.*, 1929 ; D'Elseaux and Solomon, 1933) and petit mal seizures inhibited (Lennox, 1928) by breathing mixtures rich in carbon dioxide.

The rate of cortical electrical potentials, both normal and abnormal, is greatly influenced by changes in the concentration of carbon dioxide in the alveolar air (Gibbs, Davis, and Lennox, 1935 ; Gibbs *et al.*, 1935*a*). Because changes in carbon dioxide tension have such a profound effect both on electrical

potentials and on cerebral blood flow, the inter-influence of carbon dioxide, of blood flow, and of cerebral activity need elucidation. As a result of studying the effect of various drugs on cerebral circulation and on the cortical electrical activity of the brain, we believe that although changes in action potentials of the brain may parallel circulatory changes, the two cannot ordinarily be directly correlated. In the case of carbon dioxide, however, there is a correlation the understanding of which increases greatly our knowledge of blood and brain relationships.

Data based on the concentration of carbon dioxide in the respired air or on the carbon dioxide content of blood taken from an artery or an arm vein are misleading. The carbon dioxide tension in the brain is most nearly mirrored by the carbon dioxide content of the blood from the internal jugular vein. We have observed that the sharp decrease in the carbon dioxide content of the arterial blood which accompanies hyperpnea is not followed by a decrease of similar magnitude in the carbon dioxide content of the internal jugular blood. This is because the flow of the blood through the brain has been slowed and carbon dioxide has accumulated in the venous portion of the capillaries. An opposite correction obtains when the carbon dioxide content of arterial blood is increased. Fig. 3 shows the relative carbon dioxide content of blood from an artery and from an internal jugular vein in experiments in which the gaseous content of the arterial blood was altered by over-ventilation of the lungs or by breathing increased concentrations of carbon dioxide. Each dot denotes the percentage change of carbon dioxide content in both artery and vein from the value determined before the unusual breathing took place. The chart is much wider than it is tall, which means that the changes of carbon dioxide content which occurred in arterial blood were greater than the changes in venous blood. This disproportion is most distinct in the central portion of the ordinate. Outside the central zone, changes of carbon dioxide concentration which occur in the vein are proportional to those occurring in the artery. Limitations of changes of vessel calibre and of blood pressure may limit the changes of venous carbon dioxide which occur, but another factor is the influence of the oxygen tension. Three of the points in the lower left corner of the chart are out of line. These were bloods in which the oxygen saturation in the internal jugular blood during hyperpnea was very low. As the oxygen saturation approaches 30 per cent. (and the subject approaches unconsciousness), the cerebral vessels do not continue to constrict, an observation which agrees with data obtained by other methods.

These facts indicate that the carbon dioxide tension in the pericapillary spaces is kept relatively stable. The purpose of these circulatory changes is to protect the brain from marked alterations in carbon dioxide tension. Changes in carbon dioxide tension, therefore, produce changes in the electrical activity of the brain in spite of, and not because of, cerebral circulatory adjustments.

It might be argued that the relatively high carbon dioxide content of internal jugular blood which attends hyperpnea is due not to a slower blood flow through the brain but to a transfer of carbon dioxide from the tissues to the venous blood. This point can be clarified by computing the amount of

carbon dioxide which comes from the combustion of oxygen ; in other words, by computing the respiratory quotient. The increase of carbon dioxide in the venous blood over the level in the arterial blood divided by the decrease of oxygen in the venous blood from the level in the arterial blood gives this quotient. Lennox and Leonhardt (1931*b*) ascertained that the respiratory quotient of the human brain calculated in this fashion is close to unity, 0·95,

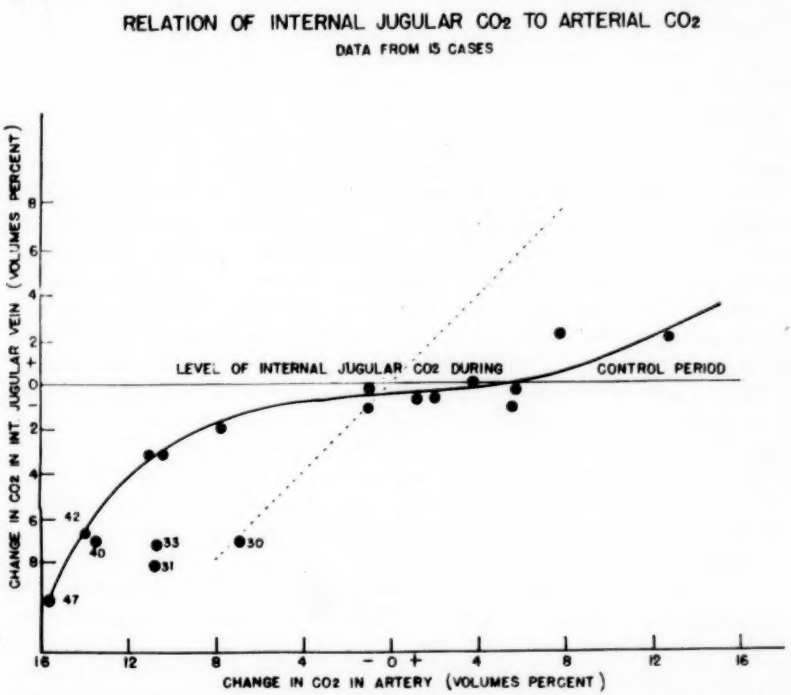

Fig. 3.—The relation of the CO_2 content of blood from an internal jugular vein and from an artery. Data are from 15 cases. Subjects " blew off " CO_2 or breathed air containing an increased concentration of CO_2. The scales represent the change in the CO_2 content of the internal jugular blood (ordinate) and of arterial blood (abscissa) from values obtained before the experiment. Each dot is the resultant of CO_2 measurements of four blood samples, i.e. the difference of CO_2 in artery and vein before and during the breathing. The figures placed beside the dots in the lower left corner are the values for oxygen saturation of internal jugular blood during hyperpnea. The dotted line indicates a one-to-one relationship.

indicating a combustion exclusively of glucose, and of the leg it is 0·87. They found that whether the subjects " blew off " carbon dioxide from the lungs or inhaled a high carbon dioxide mixture or breathed a low-oxygen mixture, the computed average respiratory quotient for the brain was the same, approximately 1·20. For the leg, however, the quotient was very different with the different procedures. When the subjects breathed 5 to 10 per cent. of carbon dioxide in air the average respiratory quotient was only 0·54 (carbon dioxide absorbed by the tissues). During hyperpnea it was 2·21 (carbon dioxide given

up by the tissues). During anoxæmia it was 2·70 (oxygen not taken up by the tissues). These data suggest that the brain does not store carbon dioxide or oxygen, and receives preferential treatment when supplies are limited.

Observations of the influence of carbon dioxide changes on the electrical activity of the cortex are even more enlightening, because definite changes in cortical potentials can be correlated with changes in mental and neural activity. We believe a frequency in cortical potentials either abnormally slow or fast, whether or not clinical symptoms are present, indicates an abnormal neurological or psychological state. Specifically, increase in carbon dioxide tension of the blood increases the frequency of the electrical waves of the brain, improves the mental activity of those with abnormally slow frequencies, and interferes with the mental activity of those whose frequencies are already normal. We have plotted frequencies of cortical oscillations against the carbon dioxide content of blood drawn from an internal jugular vein of subjects performing hyperpnea or breathing carbon dioxide. One such experiment is shown in Fig. 4. The average frequency of the electrical waves increases with increased concentrations of carbon dioxide. Therefore, we can say that within a certain normal range of carbon dioxide tension in the brain, electrical activity is exceedingly sensitive to changes in carbon dioxide tension. The effect would be much greater if the brain were not protected by the circulatory adjustments which we have discussed. Outside the usual range of normal carbon dioxide tension, cortical activity is also abnormal, but is less sensitive to further changes. It would appear, then, that normal cerebral activity is balanced on an extremely narrow base as regards carbon dioxide tension, a much narrower base than previous studies have indicated. After a certain amount of over-ventilation, the response of cortical activity varies for different persons. The cause for this individual variability requires further study.

In speaking of carbon dioxide, we emphasize that the gas is important as a participant not only in the oxidative process of cells but also in the acid-base balance of tissues.

Oxygen.—Untoward clinical effects attend oxygen pressures which are either too high or too low. In order to ascertain the degree of cerebral anoxæmia required to produce loss of consciousness, Lennox *et al.* (1935*a*) measured the oxygen saturation of blood drawn from the internal jugular vein of subjects during circulatory collapse (fainting or carotid sinus reflex with a critical fall in blood pressure), or during the breathing of nitrogen. Regardless of the cause of the collapse or of the oxygen saturation of the arterial blood, subjects were unconscious when the blood leaving the brain was less than 24 per cent. saturated with oxygen. All were conscious when the value was above 30 per cent. The critical point is, therefore, a sharp one. Patients in coma from non-circulatory causes have values of oxygen saturation well above 35 per cent.

As was stated in the discussion on carbon dioxide, alteration of the oxygen content of arterial blood does not greatly influence the cerebral circulation until extreme anoxæmia is reached. Breathing pure oxygen causes slight decrease in cerebral blood flow and breathing low-oxygen mixtures causes

increase in flow in spite of the retarding effect of the accompanying hyperpnea (Lennox and Leonhardt, 1932 ; Gibbs *et al.*, 1935*c*). When extreme, the degree of oxygen lack in internal jugular blood determines to some extent the

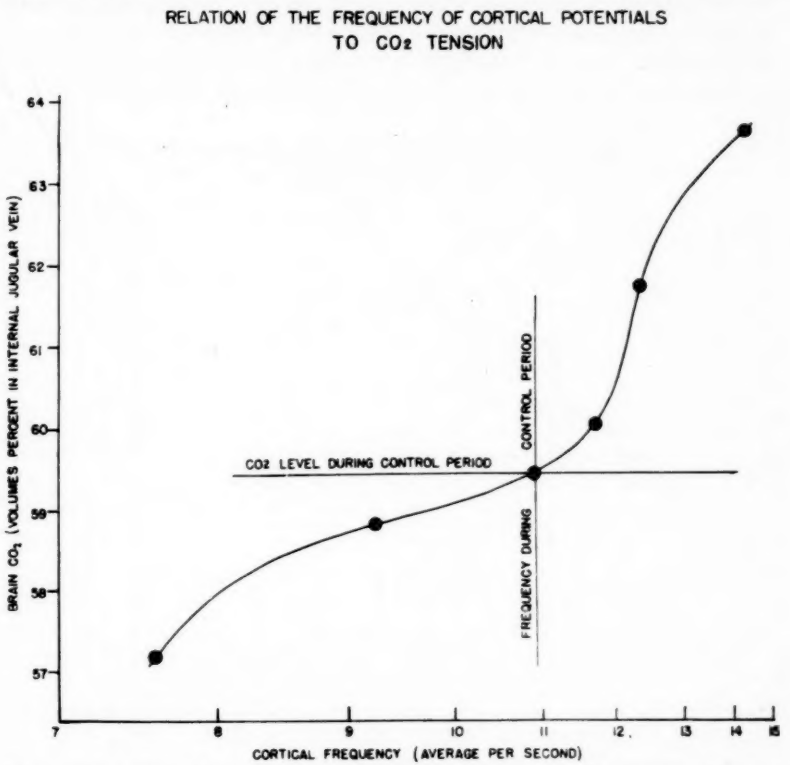

Fig. 4.—The relation of the frequency of cortical potentials to the CO_2 content of internal jugular blood ; data from one case who breathed a high CO_2 mixture or over-ventilated his lungs. The abscissa represents frequency plotted as the reciprocal of duration of wave-length, which accounts for the non-linear scale. Such a scale is desirable since a change from eight to nine per second is a greater percentage change than from 14 to 15 per second.

Average frequency per second was calculated by counting the duration of all the countable waves in a given length of electrogram from the occipital lead (indifferent electrode on the ear). The ordinate represents the CO_2 content of the internal jugular vein in volumes per cent., the best indicator of the CO_2 tension of the brain.

As can be seen, a slight change in the CO_2 content from the normal control level produces great changes in the cortical frequency, whereas when the CO_2 content is either extremely high or extremely low, the cortical frequency is less sensitive to CO_2 changes.

degree of disproportion between the carbon dioxide content of arterial and internal jugular blood (Fig. 3).

As for the effect of oxygen on cortical electrical waves, anoxæmia causes a significant slowing of frequency of the waves only when the anoxæmia is so extreme that unconsciousness impends (Fig. 5). The breathing of pure oxygen does not appreciably affect the frequency of brain rhythms. However, arterial

blood is normally nearly saturated with oxygen, and probably greatly increasing oxygen tension in the blood would increase the frequency of waves. Persons who are mildly intoxicated or are having a grand mal seizure have excessively fast cortical electrical waves (Gibbs *et al.*, 1937). Oxygen and alcoholic intoxication are each accompanied by euphoria and clumsy mental reactions, and frequently by convulsions (Lennox and Behnke, 1936).

Because moderate changes in oxygen tension have no appreciable effect on

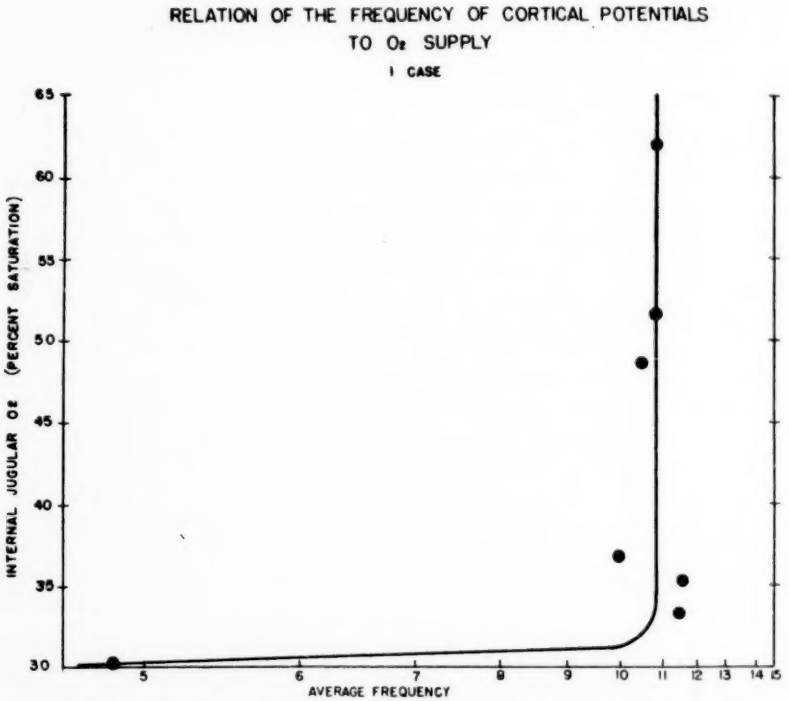

Fig. 5.—Relation of frequency of cortical potentials to the O_2 saturation of the internal jugular blood in one case. The average frequency of cortical potentials was calculated as in Fig. 4 and is represented on the abscissa scale. The O_2 saturation of blood from the internal jugular vein (ordinate) is used as an indicator of the O_2 supply of the brain. The effect of low O_2 on cortical frequency is critical, but is manifest only when the internal jugular O_2 has fallen to about 30 per cent. saturation, which approaches the level at which consciousness is lost.

the electrical activity of the cortex the cerebral circulation does not need to (and in fact does not) make adjustment for moderate changes in the oxygen content of arterial blood. The relationship of the oxygen saturation in arterial and internal jugular blood is shown in Fig. 6. For ranges of arterial oxygen which are above 70 per cent. saturation, there is no consistent relationship. Oxygen values in the blood leaving the brain are dominated by carbon dioxide values in the arterial blood. A 51 per cent. carbon dioxide value is accom-

panied by an oxygen saturation farthest to the left of the diagonal line, representing an increased cerebral blood flow ; a 42 per cent. value is farthest to the right, a decreased flow. In conditions of anoxæmia, the blood oxygen relationships are nearly linear, although, consistent with other observations, a very low arterial oxygen value is accompanied by a relatively higher internal jugular value, indicating an increased cerebral flow.

Sugar.—Moderate and temporary increase in blood sugar does not seem

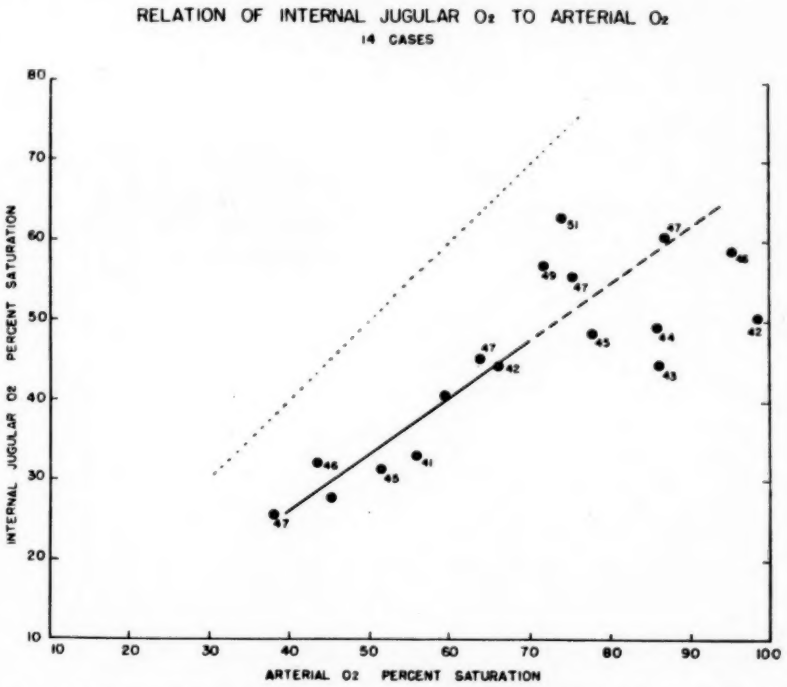

Fig. 6.—Relation of the oxygen saturation of internal jugular vein blood and of arterial blood in 14 cases in which subjects breathed air either rich or poor in oxygen. The oxygen saturations in per cent. are indicated on the ordinate for internal jugular and on the abscissa for arterial blood. The numerals beside the dots indicate the carbon dioxide content of the arterial blood during the breathing. The dotted line indicates a one-to-one relationship.

to influence normal cerebral function. Critical decrease, on the other hand, is attended by nervous and mental symptoms which are well known. Electroencephalographic observations made by Hoagland (1937) on schizophrenic patients show abnormally slow frequencies during the period of insulin reaction. Our own observations on normal subjects and on epileptics are in agreement with Hoagland's. We have, however, correlated the frequency of cortical electrical waves with the glucose content of blood, drawn not from an arm vein but from an internal jugular vein. We find that only at levels of blood

glucose below 50 mg. per cent. is the electrical activity of the cortex altered (Fig. 7).

The glucose content of the arterial blood when plotted against its content in the internal jugular blood (Fig. 8) gives a straight-line one-to-one relationship. Assuming that undue amounts of glucose are not used by the brain in hypoglycæmia, these observations indicate that the brain interposes no circulatory defence against alterations of its supply of glucose.

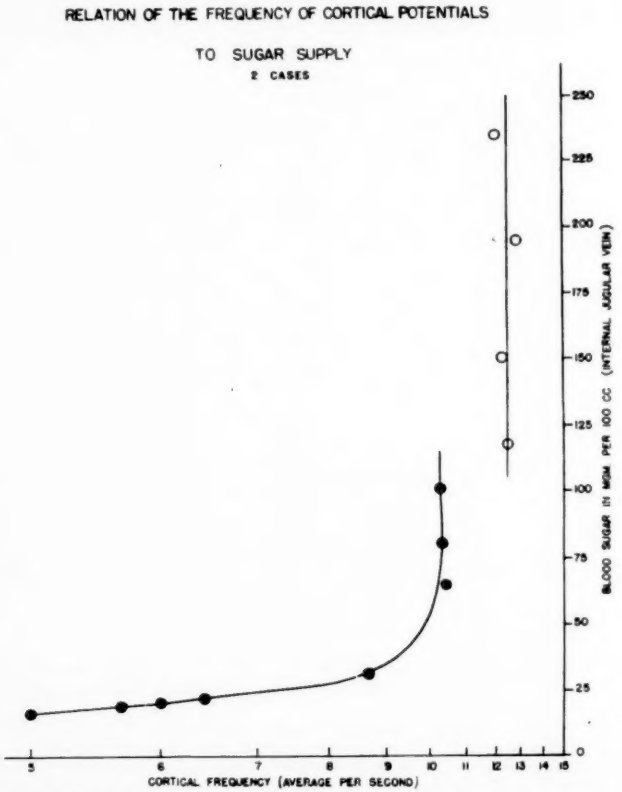

Fig. 7.—The relation of the frequency of cortical potentials to the concentration of sugar in the blood from the internal jugular vein. The data are from two cases indicated by the open and by the solid dots.

Cortical frequency (abscissa) is plotted as in Fig. 4. The amount of sugar in the internal jugular vein (ordinate) is here used as an indicator of the sugar supply to the brain. As can be seen, low sugar alters cortical frequency only at a critically low level.

Comment

Under normal conditions, the circulation in the brain is probably more constant and steady than the circulation in the arm or leg (Lennox, 1936). Unlike the muscles (except those of the heart), the brain is always active ; its functional needs are always nearly maximal and the capillaries always open.

Cerebral activity can hardly be related to such fortuitous changes in the total cerebral circulation as occur in normal activity of the brain or body. Even intermittent changes in activities, such as sleep and seizures, are initiated without benefit of gross circulatory changes. Gross changes which occur in the total cerebral blood flow are probably not adjustments to nutritional require-

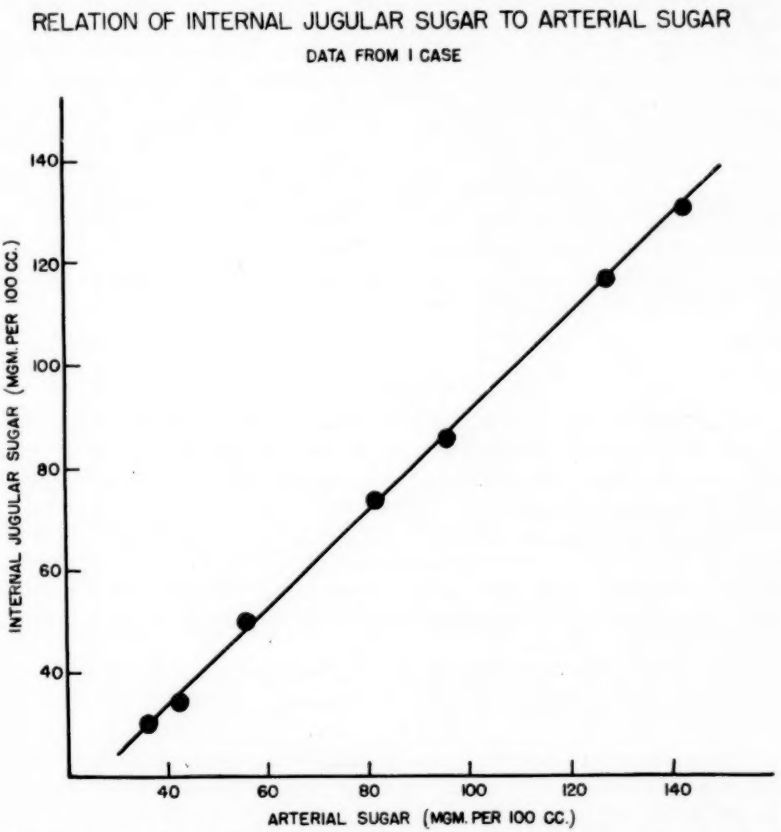

Fig. 8.—Relation of the concentration of sugar in internal jugular and in arterial blood. Arterial blood sugar measurements are plotted on the abscissa and internal jugular blood sugar on the ordinate in the case of a subject who was given insulin.

This straight-line curve indicates that there is no circulatory mechanism interposed between artery and brain to protect the brain against a low concentration of arterial sugar and that with arterial sugar levels low enough to give rise to cerebral symptoms, there is no diminution in the ability of the brain to use sugar.

ments of the nerve cells, but are directed towards a maintenance of constant carbon dioxide tensions and a constant *p*H of the nerve cells. Changes in blood flow which may occur in response to alterations in nutritional requirements are probably local.

Within the limits of clear consciousness, the greatest alterations of cerebral

blood flow attend alterations of alveolar carbon dioxide. If we consider oxygen as all-important, the action of carbon dioxide is inexplicable. For example, hyperpnea " blows off " carbon dioxide from the lungs and produces alkalosis, which in itself accentuates abnormal nervous phenomena. Yet the response of the cerebral circulation to hyperpnea is a constriction of cerebral arterioles and a stagnation of cerebral circulation. This strange action of carbon dioxide is explained when cortical action potentials are studied. Relatively slight variations in alveolar carbon dioxide influence the rate of cortical rhythms. Yet maintenance of a normal rate of the rhythms is essential for normal cerebral function. Therefore, by speeding or retarding cerebral blood flow the circulation helps to stabilize the carbon dioxide tension and the acid-base balance in the brain and allows the brain to function more normally.

In sharp contrast is the rôle of oxygen. Neither the circulation nor the electrical activity of the brain nor yet consciousness is greatly affected by variations in the oxygen concentration of the blood leaving the brain, provided the saturation is above 30 per cent. The same statement applies to glucose for concentrations above 50 mg. per cent.

Conclusions

The brain through its autonomic nervous connections should be regarded as an active rather than a passive agent in alterations of cerebral blood flow. The brain influences circulation, but within normal limits the circulation does not influence cerebral activity.

In mental activity, the total cerebral blood flow through the brain is probably increased as a result of an accumulation of carbon dioxide in cerebral tissues or because of an increase in systemic blood pressure. The increased flow presumably plays a passive and not an active part in increased mental work.

The total cerebral blood flow is not significantly decreased prior to intermittent changes in cerebral activity, such as sleep, narcoleptic and epileptic seizures.

The influence of alterations of certain blood constituents on clinical symptoms and on the electrical activity of the cortex has been studied. Increase in the carbon dioxide content of arterial blood causes a pronounced increase in the blood flow in the brain (and a decrease in the extremities) and an increase in the rate of cerebral electrical potentials. A decrease in carbon dioxide tension has the reverse effect. The alterations of cerebral blood flow serve to protect the brain against variations in carbon dioxide tension and in *p*H. With over-ventilation, for example, cerebral blood flow is slowed, and the accumulation of carbon dioxide in the capillaries increases the carbon dioxide tension in the brain and modifies the extreme decrease in arterial carbon dioxide (and the alkalosis) induced by the over-ventilation.

Moderate changes in the oxygen or the glucose content of internal jugular blood do not cause significant alterations in the cerebral circulation or in the electrical activity of the brain. Anoxæmia sufficient to affect consciousness is

attended by increased circulation and a slowing of cortical action potentials Hypoglycæmia is attended by a slowing of the electrical waves of the cortex.

Further advances in this subject will require careful analysis of the relative influence of various blood constituents and chemical processes on the metabolism and on the electrical activity of nerve cells.

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References

Behnke, A. R., Johnson, F. S., Poppen, J. R., and Motley, E. P. (1935). *Amer. J. Physiol.*, **110,** 565.
Carmichael, E. A., Doupe, J., and Williams, D. (1937). *J. Physiol.*, **91,** 186.
Cobb, S., and Fremont-Smith, F. (1931). *Arch. Neurol. Psychiat.*, **26,** 731.
Dusser de Barenne, McCulloch, W. S., and Nims, L. F. (1937). *J. cell. and comp. Physiol.*, **10,** 277.
D'Elseaux, F. C., and Solomon, H. C. (1935). *Arch. Neurol. Psychiat.*, **29,** 213.
Fulton, J. F. (1928). *Brain*, **51,** 310.
Gellhorn, E. (1937). *Amer. J. Psychiat.*, **93,** 1,413.
Gibbs, F. A., Lennox, W. G., and Gibbs, E. L. (1934). *Arch. Neurol. Psychiat.*, **32,** 257.
Gibbs, F. A., Gibbs, E. L., and Lennox, W. G. (1935*b*). *Brain*, **58,** 44.
—— (1935*c*). *Amer. J. Physiol.*, **111,** 557.
—— (1935*a*). *Amer. Heart J.*, **10,** 916.
Gibbs, F. A., Davis, H., and Lennox, W. G. (1935). *Arch. Neurol. Psychiat.*, **34,** 1,133.
Gibbs, F. A., Gibbs, E. L., and Lennox, W. G. (1937). *Arch. intern. Med.*, **60,** 154.
Hoagland, H., Cameron, D. E., and Rubin, A. M. (1937). *Amer. J. Psychiat.*, **94,** 183.
Lennox, W. G. (1928). *J. clin. Invest.*, **6,** 23.
Lennox, W. G., and Leonhardt, E. L. (1931*b*). *Arch. Neurol. Psychiat.*, **26,** 719.
—— (1931*a*). *Ibid.*, **26,** 725.
Lennox, W. G., and Gibbs, E. L. (1932). *J. clin. Invest.*, **11,** 1,155.
Lennox, W. G., Gibbs, F. A., and Gibbs, E. L. (1933*a*). *Arch. Neurol. Psychiat.*, **34,** 1,001.
Lennox, W. G., Gibbs, E. L., and Gibbs, F. A. (1935*b*). *J. Pharmacol.*, **53,** 113.
Lennox, W. G., and Behnke, A. R., Jr. (1936). *Arch. Neurol. Psychiat.*, **35,** 782.
Lennox, W. G., and Gibbs, E. L. (1936). *Ibid.*, **36,** 13.
Lennox, W. G. (1936). *Ibid.*, **36,** 375.
Lennox, W. G., Gibbs, F. A., and Gibbs, E. L. (1936*a*). *Ibid.*, **36,** 1,236.
Lennox, W. G., and Williams, D. (unpublished observations).
Loevenhart, A. S., Lorenz, W. F., and Waters, R. M. (1929). *J. Amer. med. Ass.*, **92,** 880.

OBSERVATIONS ON THE CEREBROSPINAL FLUID PRESSURE ON SIMULTANEOUS VENTRICULAR AND LUMBAR PUNCTURES

BY

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(RECEIVED 28TH APRIL, 1938).

THE investigations which form the basis of this report were undertaken with the object of determining the relations existing between the ventricular and lumbar cerebrospinal fluid pressures. In the past it seems to have been taken for granted that the pressure within the cerebral ventricles is always in equilibrium with the pressure throughout the entire extent of the spinal subarachnoid space, except, of course, in the presence of spinal block. From time to time, however, it has been suggested that obstruction at the foramen magnum by herniation of the cerebellar tonsils in cases of intracranial tumour might possibly cause a state of partial block. This suggestion has generally been advanced in explanation of the not unusual finding of a normal, or even subnormal, lumbar pressure despite the presence of clinical signs indicative of high intracranial pressure.

Hodgson (1929) reported a series of 49 cases of intracranial tumour which he had investigated by a method described as "combined ventricular and lumbar puncture." He concluded that partial block might result from herniation of the cerebellar tonsils, and he went so far as to say that under such circumstances the initial lumbar pressure might be lower than the pressure in the ventricles. The method he used was as follows. The ventricle was tapped and the ventricular pressure measured. If this pressure was 200 mm. of water or more, cerebrospinal fluid was allowed to escape until the pressure was reduced well below this level. Lumbar puncture was then carried out ; the lumbar pressure was measured and, finally, the responses on jugular compression were noted. It will be apparent that the limitations imposed by this method did not allow a determination of the initial simultaneous lumbar and ventricular pressures, and, in fact, Hodgson did not publish any actual figures of the pressure relations in his cases. Furthermore, he used a manometer tube with a bore of 2·0 mm. in measuring the ventricular pressure, and in such a tube a column of fluid 300 mm. in length has a volume of about 1·0 c.c. The displacement of this quantity of fluid is of itself sufficient to cause a variable but definite lowering of the ventricular pressure.

The methods used in the investigations here reported are believed to be free

from these possibilities of error, and the results lead us to conclude that in certain cases of supratentorial tumour the ventricular pressure is greater than the lumbar. Further, it is highly probable that this difference in pressures is causally related to herniation of the cerebral hemisphere through the incisura tentorii.

Method

An initial ventricular tap was carried out in the usual manner. The ventricle chosen was the one which, on clinical grounds, was considered to be on the unaffected side. In every case the operation was carried out under local infiltration anæsthesia. Premedication was not employed. The tap was made with a needle specially designed to guard against loss of fluid and to facilitate the attachment of a manometer tube. Essentially it was a Greenfield lumbar puncture needle, but with a blunt end and the eye laterally placed. When the needle was judged to be within the ventricle the stylet was cautiously withdrawn until fluid was seen to well up around it. At this point the tap was closed and the patient was turned into a lateral position. Lumbar puncture was next carried out, care being taken to avoid loss of fluid. Manometer tubes were then attached to both needles. They were of glass, 300 to 400 mm. in length, and with a uniform bore of 1·0 mm. When necessary the manometer tube was easily extended by adding a second length of tubing by means of a short rubber connector. At this stage the position of the patient and operation table were adjusted in order to bring both needles into the horizontal plane. A piece of board some 3 ft. long and a spirit-level were found convenient for making this adjustment and, as a final check, a water U-tube was used, consisting of two glass tubes connected by several feet of thick rubber tubing.

The pressure relations were investigated in a uniform manner, the various steps of which are conveniently shown in the following tabulation :

(i) Separate measurement of the lumbar and the ventricular pressures.

(ii) Both taps opened and measurement of the two columns of fluid. If any difference was noted the position of the needles and menisci was carefully checked with the spirit-level and water U-tube.

(iii) Bilateral jugular compression, noting the changes effected first in each manometer opened separately and then in both simultaneously.

(iv) Withdrawal of a measured amount (usually 5·0 c.c.) of cerebrospinal fluid from the lumbar puncture.

(v), (vi), and (vii) Repetition of steps (i), (ii), and (iii).

In a few instances the critical condition of the patient necessitated a curtailment of the investigations, but in nearly every case the first three steps at least were carried out.

It is, perhaps, as well to emphasize that a column of fluid 300 mm. in length in a tube with a bore of 1·0 mm. represents about 0·25 c.c. of fluid, and the withdrawal of this quantity is insufficient to cause appreciable lessening of the ventricular or lumbar pressures.

Results

Forty-two observations were made on 39 patients. In the accompanying table the cases are classified according to the pressure of the cerebrospinal fluid and the character of the responses on jugular compression.

TABLE I

GROUPS	TOTAL CASES	SITUATION OF TUMOUR		DEATHS
		SUPRATENTORIAL	SUBTENTORIAL	
I : Normal	6	—	—	—
II : Tumours with normal pressures	5	5	—	1 (20%)
III : Tumours with raised pressure and normal dynamics	20	15	5	6 (30%)
IV : Tumours with abnormal dynamics	8	6	2	6 (75%)
Total cases with tumour ..	33	26	7	13 (39·3%)

Group I.—The six patients in the first group were submitted to ventriculography on the suspicion that a space-occupying intracranial lesion was present. Traumatic neurosis, cortical atrophy, and late epilepsy were some of the alternative diagnoses. All the patients were alike in that there was absence of clinical signs of increased intracranial pressure, and in all the cerebrospinal fluid pressures were within the range commonly accepted as normal. One patient, suffering from a traumatic neurosis, had a lumbar pressure of 170 mm. of water; in the others the pressures varied between 100 and 145 mm. of water.

In all the patients of this group the initial lumbar and ventricular pressure were equal and the responses on jugular compression were prompt, synchronous, and of equal range.

Group II.—The five patients in the second group all had intracranial tumours, proved at operation. The highest cerebrospinal fluid pressure recorded in this group was 185 mm. of water; this was in a man who had a left parietal meningioma weighing 130 gm. The lowest pressure recorded was 90 mm. of water in a patient who had a deeply placed right temporal cystic glioma.

It is of some interest that three of the five patients of this group had supratentorial meningiomas which were successfully removed.

The initial ventricular and lumbar pressures were equal, and they remained equal through the various stages of the test. The responses on jugular compression were prompt and of equal range and on release the return to the resting level was immediate.

Group III.—The 20 patients in the third group all had intracranial tumours and in them the cerebrospinal fluid pressures were abnormally high (200 mm. of water or more). The raised cerebrospinal fluid pressure constituted, however,

the sole abnormality of the cerebrospinal hydrodynamics, for the ventricular and lumbar pressures were equal in all the circumstances imposed by the tests employed. Particular importance attaches to the five examples of subtentorial tumour in this group. In four of these five cases there was either operative or post-mortem verification of the tumour, and in all four herniation of the cerebellar tonsils was very great (Fig. 1). Nevertheless, this herniation through the foramen magnum was not accompanied by any inequality of the lumbar and ventricular pressures, neither did it cause any retardation in the responses to jugular compression.

Of the 15 examples of supratentorial tumour, three were examined post mortem and in two of these three some degree of tonsillar herniation was present ; in only one of them was there any post-mortem evidence of tentorial herniation, and it was of the slightest degree. Moreover, the patient survived the investigation by rather more than three weeks, and it is at least possible that the tentorial herniation occurred in the last weeks of life.

Group IV.—The eight patients comprising the fourth and important group are those who showed some abnormality of the cerebrospinal hydrodynamics apart from a simple increase of pressure. The essential data relating to these cases are set out in Table 2.

Two patients (Cases 7 and 8) showed a relatively slight abnormality, which consisted of a definite retardation in the lumbar response to bilateral jugular compression. In the other six patients an initial difference in the lumbar and ventricular pressures was found, and, except in Case 2, this more striking abnormality was always accompanied by a marked retardation in the lumbar response to jugular compression.

In the six patients in whom the ventricular pressure was initially greater than the lumbar, the disparity varied from 20 mm. to as much as 100 mm. of water. In all cases except Case 4, in which only 2 c.c. of fluid were removed, the withdrawal of fluid from the lumbar sac accentuated the disparity. Only one of these six patients had a subtentorial tumour, and this patient had bilateral auditory neurofibromata.

In all five cases where the tumour was supratentorial, there was clear post-mortem proof of herniation of the ipsilateral temporal lobe through the incisura tentorii (Fig. 2), and of a greater or less degree of pressure upon the contralateral crus (Kernohan and Woltmann's notch). In this connection it is perhaps as well to point out that the tonsillar pressure cone is much more easily demonstrated post mortem than the tentorial herniation. The post-mortem technique used at the National Hospital (the cadaver in the prone position with the head flexed over a block) permits an excellent exposure of the contents of the posterior cranial fossa and upper cervical vertebral canal, and shows the disposition of the parts *in situ*. Thus, even slight degrees of tonsillar herniation are at once apparent. There is, unfortunately, no means of obtaining a comparable exposure of the region of the incisura tentorii. Here one must depend solely on the appearances after removal of the brain, and it is highly probable that tentorial herniations become less evident, just as is the case with the tonsillar herniation.

TABLE II.—CASES SHOWING

CASE	DISEASE	PRESSURES BEFORE WITHDRAWAL OF FLUID (MM. OF WATER)					FLUID REMOVED FROM LUMBAR PUNCTURE
		MEASURED SEPARATELY		MEASURED SIMULTANEOUSLY		BILATERAL JUGULAR COMPRESSION	
		LUMBAR	VENTRIC.	LUMBAR	VENTRIC.		
1. J. C., 37 ..	L. frontal glioma (cystic).	280	310	240	300	Lumbar: slow rise to 300. Ventric.: quick rise to 480.	5 c.c.
2. F. M., 30	L. temporal lobe abscess (otitic).	360	450	350	450	Lumbar: fairly quick rise to 450. Ventric.: quick rise to 600.	5 c.c.
3. L. W., 31	R. parietal glioma.	385	430	—	—	Lumbar: slow rise. Ventric.: prompt rise.	5 c.c.
4. F. R., 40..	Large middle fossa meningioma.	330	370	310	330	Lumbar: slow rise to 400. Ventric.: quicker rise to 500.	2 c.c.
5. M. D., 29	R. occipital glioma.	490	460	340	360	Lumbar: slow rise. Ventric.: quicker rise.	5 c.c.
6. M. C., 29	Bilateral VIII N. neurofibromata.	320	340	300	320	Lumbar: slow rise to 350. Ventric.: quick rise to 420.	5 c.c.
7. A. C., 44	R. parietal tumour.	345	345	—	—	Lumbar: slow rise to 360. Ventric.: quick rise to 400.	5·2 c.c.
8. H. B., 65	Posterior fossa tumour (unverified).	95	95	—	—	Lumbar: slow rise to 150. Ventric.: quick rise to 300.	3 c.c.

Note.—In Case 5 it will be observed that the initial lumbar pressure was higher than pressure became greater than the lumbar, and this excess of ventricular pressure persisted doubt that the latter readings were a true index of the pressure relations.

ABNORMAL DYNAMICS.

PRESSURES AFTER WITHDRAWAL OF FLUID (MM. OF WATER)					EXCESS OF VENTRIC. PRESSURE		REMARKS
MEASURED SEPARATELY		MEASURED SIMULTANEOUSLY		BILATERAL JUGULAR COMPRESSION	BEFORE WITHDRAWAL	AFTER WITHDRAWAL	
LUMBAR	VENTRIC.	LUMBAR	VENTRIC.				
120	220	140	210	Lumbar: slow rise to 170. Ventric.: quick rise to 340.	30	100	Died same day. Tentorial and tonsillar herniation.
210	(?) patient restless.	—	—	—	100	—	Died within 24 hours. Severe tentorial herniation but no tonsillar herniation.
230	285	—	—	Block more evident.	45	55	Died same day. Tentorial and tonsillar herniation.
270	300	270	300	As before.	40	30	Death 3 weeks later. Severe obstruction at the tentorium. No tonsillar herniation.
200	250	200	250	As before.	20	50	Death within 24 hours. Severe tentorial herniation, no tonsillar herniation.
220	260	240	270	As before; indication of block more marked.	20	40	Died 2 days later. Large tumours filling both cerebello-pontine angles. No tonsillar herniation.
140	140	—	—	Lumbar: very slow rise to 160. Ventric.: quick rise to 400.	—	—	Discharged from hospital I.S.Q.
30	30	—	—	As before.	—	—	Discharged from hospital I.S.Q.

the ventricular. However, when both taps were open simultaneously the ventricular and was increased after withdrawal of fluid by the lumbar route. We have little

Cases 7 and 8 showed merely a retardation in the rise and fall of the lumbar pressure on jugular compression. Both patients were discharged from hospital and final verification of the tumours is lacking. In Case 7 the clinical findings and the exploration of the right cerebral hemisphere gave almost certain proof of the existence of a deep temporo-parietal tumour. Case 8 was an old and feeble woman ; following ventriculography a suboccipital decompression, without exploration, was carried out for the relief of the high intracranial pressure.

Discussion of Results

It is apparent that the observations here recorded afford conclusive proof that the lumbar and ventricular pressures are not of necessity always equal. A difference was observed only in cases where the lumbar pressure was abnormally high. It seems, therefore, that a normal lumbar pressure in a case of intracranial tumour is a true index of the intracranial pressure. In Case 8 the patient had an acute papillœdema of three dioptres, and the lumbar pressure, although only 90 mm. of water, was identical with the ventricular pressure.

Although the maximum difference of pressure did not exceed 100 mm. of water, the clinical course of the patients indicates that a considerably smaller difference of pressure is of grave significance.

The conditions determining the occasional inequality of pressures are worthy of further consideration. Of 33 patients suffering from intracranial tumour, there were six in whom the ventricular pressure exceeded the lumbar. In five of these six patients the tumour was situated above the tentorium, and without exception there was definite post-mortem evidence of herniation of the ipsilateral temporal lobe through the incisura tentorii. In contrast with these observations are the findings in four patients suffering from proved subtentorial tumour. In them, despite marked and in some cases extreme tonsillar herniation, the lumbar and ventricular pressures were equal under all the conditions imposed by the several tests employed. Furthermore, the lumbar and ventricular responses to bilateral jugular compression were equal in all respects.

When these investigations were begun it seemed reasonable to anticipate that herniation through the foramen magnum might cause some degree of block. The normal pressure relations in cases of subtentorial tumour which were associated with severe tonsillar herniation came, therefore, as something of a surprise, particularly as certain very abnormal observations were recorded in cases of supratentorial tumour. Case 8, a patient with an unproved though almost certain subtentorial tumour, showed, it is true, a distinct lag in the rise of lumbar pressure on jugular compression, and it may be that had a greater number of cases of subtentorial tumour been examined more would have shown some degree of abnormality of the cerebrospinal hydrodynamics other than a simple increase of pressure. Two factors, however, reduced the number of patients with subtentorial tumour available for study. First, a large proportion of subtentorial tumours occur in children, with whom co-operation is difficult or impossible, and secondly, in subtentorial tumours in adults ventriculography is not often necessary.

The only proved case of subtentorial tumour showing a difference in the

lumbar and ventricular pressures was a patient with large bilateral auditory neurofibromata. In addition, there were numerous small neurofibromata on the posterior spinal roots. None of these was sufficiently large to have caused a block, and we believe that the inequality in the lumbar and ventricular pressures must be related to the auditory nerve tumours. However, there is obviously a great difference between a unilateral neurofibroma or a midline cerebellar tumour and tumours filling up both cerebello-pontine angles. Indeed, it is plain that the mechanical effects produced by the obliteration of both angles, together with probable coincident compression of the iter of Sylvius, would not be far removed from those brought about by herniation of the temporal lobe downwards through the incisura tentorii. For these reasons the observations in this case can hardly be advanced as evidence in favour of tonsillar herniation being a usual cause of fluid block.

Assuming that tentorial herniation stands in causal relation with a ventricular pressure in excess of the lumbar, it is advantageous to consider the manner in which such a disparity in pressures is likely to be produced. The factors in operation can best be illustrated by considering the probable stages in the evolution of a unilateral supratentorial tumour. In the earlier stages of the growth of such a tumour, the affected cerebral hemisphere increases in bulk at the expense of the adjacent subarachnoid cisterns. As Spatz and Stroescu (1934) have shown, this is at first, in all probability, a passive process, but at a later stage the ever-increasing demand for space leads to complete obliteration of the cisterns and, particularly, of the cisterna ambiens (the subarachnoid cistern encircling the mid-brain). The obstruction in this situation corresponds with that experimentally produced in dogs by Dandy (1919). He found that the production of adhesions around the mid-brain invariably led to the development of hydrocephalus. The obliteration of the cisterna ambiens, on the basis of Dandy's experiments, affords a reasonably adequate explanation for a general increase of intracranial pressure, but the resulting obstruction to the upward flow of cerebrospinal fluid does not, of course, account for a disparity of pressures above and below the tentorium. The increasing bulk of the cerebral hemisphere, however, would serve to force the medial border of the temporal lobe, lying as it does immediately above the incisura, downwards through the incisura tentorii. In this way a frank tentorial herniation is produced, and as it progresses it is likely that the iter of Sylvius becomes obliterated, or at least so compressed that it is inadequate to equalize the pressures above and below the tentorium. In such circumstances the pressure in the posterior cranial fossa is not necessarily the same as the pressure above the tentorium, and it may well be lower because of possible absorption of cerebrospinal fluid below the tentorium. In our opinion this would explain the occurrence of a lumbar pressure which is lower than the pressure in the ventricles. In short, it is suggested that obliteration of the cisterna ambiens is the cause of the simple increase of intracranial pressure observed in the cases comprising Group III, and that a superadded compression of the iter of Sylvius is the factor responsible for the disparity of pressures which was found in the cases in Group IV.

The observations made in cases of subtentorial tumour with severe tonsillar herniation give strong warrant for the belief that the lumbar pressure is identical with the pressure in the posterior cranial fossa, and therefore justify the assumption that lumbar and ventricular punctures give a true indication of the pressures below and above the tentorium. It should be noted, however, that the pressure in the cisterna magna was not measured in any of the cases. Cisternal puncture is hardly justifiable where there is a suspicion of raised intracranial pressure.

These observations warrant some further consideration because of the bearing they have on prognosis and treatment. An inspection of Table I shows that the mortality rate was 30 per cent. in Group III, in which the only abnormality was a raised intracranial pressure, whereas it was 75 per cent. in Group IV, in which an additional abnormality in the cerebrospinal hydrodynamics was present. In Group IV, of the six patients with a difference of lumbar and ventricular pressures, four died within 24 hours of investigation. In three of the cases (Cases 1, 3, and 5), this rapidly fatal issue followed a simple decompression and in the fourth case (Case 2) after nothing more than a ventricular tap. It is highly probable that the tentorial herniations were largely responsible for the early death in these four cases. In the other two cases death was delayed ; in Case 6 it occurred 2 days later from the severity of the operation, and in Case 4 after 3 weeks from the natural course of the disease. This mode of death is in marked contrast with that in the six patients in Group III, in whom the only abnormality of the cerebrospinal hydrodynamics was a raised intracranial pressure. Of these six patients all but one survived operation by more than 3 days, and death was due to the natural progress of the disease. The early death on the second day in one patient was due to the effects of a radical tumour removal.

This study of the mortality figures for the several groups, even though based on relatively few cases, suggests the gravity of a tentorial herniation. While it is probable that the sudden death which has long been known to occur in cases of unrelieved cerebellar tumour is due to medullary compression associated with tonsillar herniation, the gravity of tentorial herniation has only recently been recognized. In this connection it is interesting to consider certain collateral evidence which has recently accumulated.

Herniation of the brain through the incisura tentorii and the foramen magnum was well known to Collier (1904), and the existence and significance of notching of the crus was pointed out by Kernohan and Woltmann (1929). These writers, however, were principally concerned with the false localizing signs which accompanied these conditions. Clovis Vincent, David and Thiébaut (1936) appear to have been the first to stress the great danger of tentorial herniation, and David and Askenasy (1937) gave a graphic account of the clinical course in such cases. They stressed the danger of lumbar puncture and urged that ventricular puncture was a procedure of much greater safety. Even ventricular puncture was, in their opinion, by no means devoid of risk, and they advised that it should only be done as an immediate preliminary to operation. Finally, they urged the importance of searching for the tentorial herniation

during operation and, if possible, disimpacting it. In two of their cases where rapidly increasing coma followed on radical operation for supratentorial tumour, they re-explored the brain and successfully liberated a herniated temporal lobe. The clinical course of the cases with tentorial herniation here reported bears out their contentions. Furthermore, withdrawal of cerebrospinal fluid accentuated the disparity between the lumbar and ventricular pressures, and the implication of this requires no emphasis. In the experience of one of us (W. R. H.) ventriculography, when not followed by immediate operation, has in several instances proved fatal within 24 to 48 hours. In these cases the tumours were supratentorial and it appears that the risk of ventriculography, contrary to what is often stated, is much greater in cases of supratentorial tumour than it is in the presence of hydrocephalus caused by subtentorial tumour.

Massive ponto-mesencephalic hæmorrhages, which are known to occur in the presence of supratentorial tumours when associated with tentorial herniation, are of considerable significance when considered in the light of the present observations. Van Gehuchten (1937) reported six cases, and in all of them the tumours were supratentorial and the degree of tentorial herniation severe. In four of the cases death appeared to follow lumbar puncture or ventriculography. He was in entire agreement with David and Askenasy regarding the dangers attendant on lumbar puncture. Moore and Stern (1938) collected 10 cases of massive ponto-mesencephalic hæmorrhage in 130 consecutive cases of intracranial tumour examined post mortem at the National Hospital. The presence or absence of tentorial herniation was not stated in every case, but it was present in the majority. In all the 10 cases the hæmorrhage appeared as a terminal event and it had no specific connection with lumbar puncture, ventriculography, or operation. The character of these hæmorrhages was remarkably constant. In all cases they were arterial or at least arteriolar, and occurred chiefly from the long central ascending branches of the basilar artery. Van Gehuchten and Moore and Stern all sought the reason for the occurrence of the hæmorrhages in the increased intracranial pressure, and they suspected that tentorial herniation played an important part in their causation.

It appears to us that a disparity in the intracranial pressures above and below the tentorium may partly explain the occurrence of ponto-mesencephalic hæmorrhage, for the conditions which appear to determine the hæmorrhage are exactly those which were found in our cases to be associated with a difference of lumbar and ventricular pressures. At first sight it appears strange that no instance of ponto-mesencephalic hæmorrhage was met with in the present series, but it is to be remembered that of the six cases in Group 4 who showed a disparity of pressures, all but one had some form of decompression. Some of the recorded cases of ponto-mesencephalic hæmorrhage, on the other hand, had ventriculography which was not as a rule followed by operation. The hæmorrhage, in fact, appeared as a terminal event in cases of unrelieved high intracranial pressure. It appears that a continued excess of ventricular over subtentorial pressure is a factor the importance of which should not be ignored in any hypothesis which seeks to explain the occurrence of these ponto-mesencephalic hæmorrhages.

If the intracranial circulation is to remain adequate in states of increased intracranial pressure, some adjustment of the general arterial pressure must occur to meet the requirements of the raised intracranial pressure. Assuming that the arterial pressure is uniform throughout the intracranial cavity, an arterial pressure which is adequate for the region of highest intracranial pressure would presumably be excessive in a region of relatively lower intracranial pressure. When the intracranial pressure is lower below than above the tentorium, there will be an excessive difference between the arterial and the cerebrospinal fluid pressures in the posterior cranial fossa, and when this difference becomes sufficiently great it will cause stretching of the arterial wall and hæmorrhage ; and, indeed, Moore and Stern point out that the pathological features of ponto-mesencephalic hæmorrhage complicating supratentorial tumour are identical with those seen in ordinary hypertensive apoplexy.

It is suggested, therefore, that ponto-mesencephalic hæmorrhages occurring in cases of supratentorial tumour are the result of a relative arterial hypertension, which is due to the fact that the intracranial pressure is lower in the posterior cranial fossa than it is above the tentorium.

Summary and Conclusions

The relationship between the lumbar and ventricular cerebrospinal fluid pressures has been investigated on 41 occasions in a series of 39 patients, the majority of whom were suffering from intracranial tumour.

Six of the patients had no gross intracranial disease, and in them the lumbar and ventricular pressures were identical when measured separately, simultaneously, on bilateral jugular compression, and after withdrawal of lumbar fluid.

All the other patients, except one, had proved intracranial tumours. In five of them the initial lumbar pressure was within normal limits and in 20 the lumbar pressure was above 200 mm. of water; in all 25 cases the lumbar and ventricular pressures and the responses to jugular compression and after withdrawal of fluid were identical. In the remaining and important group of eight cases, six showed an initial difference between the lumbar and ventricular pressures, the ventricular pressure being from 20 to 100 mm. higher. This disparity was accentuated by withdrawal of lumbar fluid, and in all eight cases jugular compression revealed a partial block.

A difference between the lumbar and ventricular pressures was associated with a high early operative mortality and with post-mortem evidence of herniation of the temporal lobe through the incisura tentorii. Tonsillar herniation through the foramen magnum, on the other hand, did not produce any difference of pressures.

The significance of these findings is discussed as regards the importance of tentorial herniation in relation to the possible dangers of lumbar puncture and ventriculography, and as an important factor in causing early post-operative death. Furthermore, the occurrence of tentorial herniation, associated with

an inequality of supratentorial and subtentorial pressure, is believed to be an important factor in causing the ponto-mesencephalic hæmorrhage which may occur as a terminal event in cases of cerebral tumour with unrelieved high intracranial pressure.

It is a pleasure to acknowledge our thanks to Dr. E. Arnold Carmichael and Dr. J. G. Greenfield for their interest and criticism, and to the Honorary Medical and Surgical Staff of the National Hospital for allowing us to make these observations on their patients. One of us (G. E. S.) held a Dickinson Research Scholarship.

REFERENCES

Collier, James (1904). *Brain*, **27,** 490.
Dandy, W. E. (1919). *Ann. Surg.*, **70,** 129.
David, M., and Askenasy, H. (1937). *J. Belge Neurol. Psychiat.*, **37,** 550.
Van Gehuchten, P. (1937). *Encéphale*, **32,** ii, 113.
Hodgson, J. S. (1927). " The Intracranial Pressure in Health and Disease " : The Association for Research in Nervous and Mental Disease, **8,** 182.
Kernohan, J. W., and Woltmann, H. W. (1929). *Arch. Neurol. Psychiat.*, Chicago, **21,** 274.
Moore, M. T., and Stern, K. (1938). *Brain*, **61,** 70.
Spatz, H., and Stroescu, G. J. (1934). *Nervenarzt*, **7,** 425.
Vincent, Cl., David, M., and Thiébaut, F. (1936). *Rev. Neurol.*, **65,** 536.

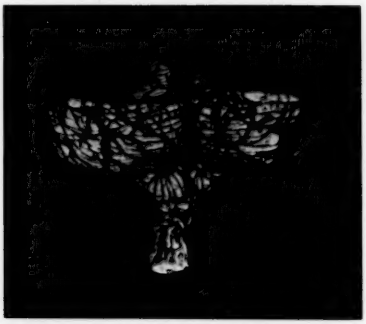

Fig. 1.—Posterior view of cerebellum showing downward herniation of the cerebellar tonsils

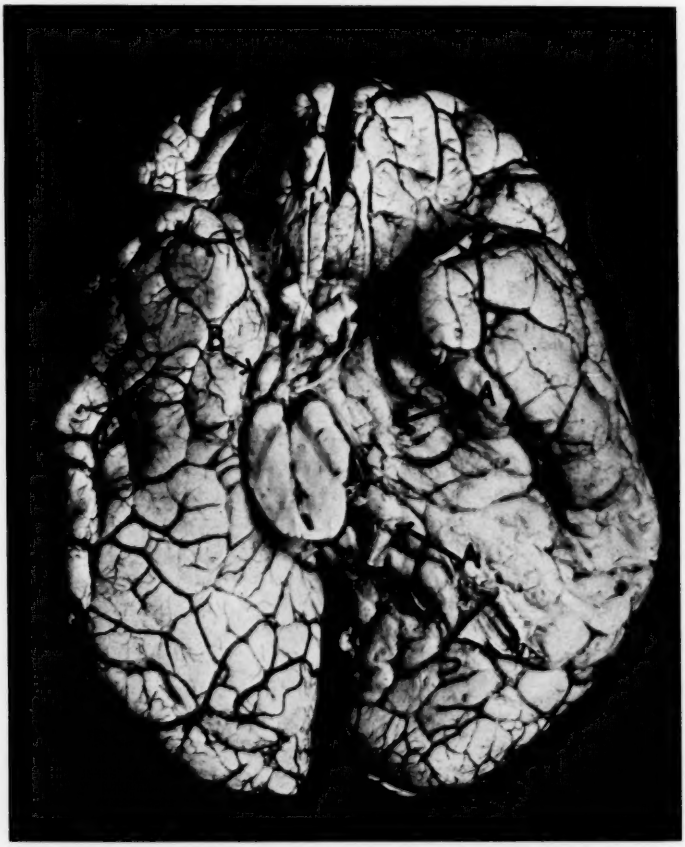

Fig. 2.—The brain from a case of left temporal abscess (Case 2) showing a severe tentorial herniation and dislocation of the midbrain to the opposite side. The impression of the edge of the tentorium is indicated by the arrows A. Note the slight accentuation of the normal groove (B) on the right uncus.

A CRITICAL REVIEW

TWIN RESEARCH IN PSYCHIATRY

BY

E. T. O. SLATER

General Principles and Theoretical Basis

IT is not attempted in the following review to cover the whole of the now enormous literature on twin research, even as far as it concerns psychiatry. Only works of statistical or theoretical importance will be dealt with in a critical spirit. Extensive reviews of twin literature from the general aspect are given by Newman (1923), Siemens (1924), Dahlberg (1926), von Verschuer (1927), Siemens (1932), von Verschuer (1932), Luxenburger (1932*c*), von Verschuer (1933), Newman, Freeman, and Holzinger (1937). Reviews of the literature as far as it specially affects psychiatry are given by Siemens (1924), Lange (1929), Luxenburger (1929, 1930*a* and *b*, 1932*a*, 1933, 1935), and Conrad (1937). No account will be given of the now very large number of papers dealing with work on twins of purely psychological nature. It is, however, time that these works, which are of very varying merit, were submitted to critical consideration by a competent reviewer.

Twin research in psychiatry, as in other fields of human biology, is founded on a particular theory, that there are two kinds of twins, the monozygotic and the dizygotic. Both members of a pair of monozygotic twins are supposed to have exactly the same hereditary equipment. The members of a pair of dizygotic twins are supposed to be no more like each other than ordinary siblings. If this theory is correct, we are enabled to observe the results of a controlled experiment performed by nature. For if the hereditary endowment is the same, any real differences observed in monozygotic twins should be due to the environment. The differences in dizygotic twins will be due to differences both in the environment and in the hereditary equipment. This theory is probably correct (though it cannot be taken as proven), although its theoretical embryological basis has become more dubious and speculative than it seemed at first.

The distinction between monozygotic and dizygotic twins was originally based on the observation that some twins were born with separate placentæ and membranes, some with these structures in common. The latter variety were found to be always of the same sex, and as they grew up they grew in-

creasingly like one another, so like in fact that it was often difficult to distinguish them. From a statistical point of view it was found that there was an excess number of twins born of like sex, and were one to deduct double the number of opposite-sexed twins from the total, the remaining proportion represented about the same figure as the proportion of monochorial or of similar twins. Further, it has been found that this group of similar twins, if selected on grounds of their similarity to one another in a number of characteristics commonly held to be hereditary (such as eye colour, stature, etc.), would be found to be similar in a vast number of other hereditary characteristics. A similar phenomenon would not be observed in a group of similarly chosen opposite-sexed twins. It was clear, therefore, that this group of twins formed a special group in which the hereditary constitution could be taken to be very nearly if not absolutely the same in both members of the pair. For a very long period the diagnosis of these two groups was by means of the membranes only. As, however, the placental membranes of adult twins are not easily to be rescued from the incinerator to which they have many years before been consigned and as any reliable history of the formation of those placentæ is often quite unobtainable, the method of diagnosis by similarity has gradually replaced the diagnosis by membranes, and is now to be regarded as the method of choice. For the diagnosis by membranes, even when carried out with the most exact care, can no longer be regarded as reliable. Smith (1930 and 1931) has described almost certainly uniovular dichorial twins and Curtius (1930) and Lassen (1931) have made a systematic examination of the point. Between them they examined 86 twin placentæ by macroscopical, microscopical, and Roentgenological methods, and carried out a very careful anthropological investigation of the like-sexed twins who could be followed up. Lassen gives their results in the following table :

	NO. OF CASES	PLACENTÆ : 1	2	ANASTOMOSES		SIMILARITY DIAGNOSIS BY ANTHROPOLOGICAL METHODS
				MACROSCOPIC	SEEN IN X-RAY ONLY	
Monochorial ..	9	9	—	7	1	Similar.
Dichorial ..	21	16	5	—	—	Opposite sex, dissimilar.
„ ..	21	9	12	—	—	Like sex, dissimilar.
„ ..	5	2	3	—	1	Similar.

They were unable to find any biological difference between the monochorial and the dichorial similar twins, from which it follows that the presence of one or two choria is more or less an accidental phenomenon and without much significance for the diagnosis of constitutional similarity or dissimilarity. As they found among 14 similar twins no less than five dichorial pairs, they conclude that dichorial uniovular twins are not at all uncommon ; and this opinion is borne out by the fact that the percentage of monochorial twins has been per-

sistently found to be smaller than the percentage of uniovular twins calculated from the sex distribution. Curtius (1930) considers that his results indicate that the method of diagnosis by membranes is unreliable and should be discarded. The occurrence of monochorial dizygotic twins appears to be much less common, if it occurs at all. Von Verschuer (1932*a*) quotes two cases of opposite-sexed monochorial twins from the literature, and has himself described (1925) a case of monochorial dizygotic same-sexed twins. The evidence for monochoria rests on the statement of an obstetrician. In view of the rarity of this phenomenon, even if it does occur, an exact diagnosis of monochoria, supported by microscopical examination, must be taken as very strong evidence of uniovularity. There is no particular difficulty from the embryological point of view in explaining the occurrence of dichorial uniovular twins, as long as the point of separation of the twin embryos is considered to be capable of occurring before the development of the chorionic anlage. Siemens (1924), who was to a large extent the author of the modern method of diagnosis by similarity, has stigmatized the diagnosis of ovularity by membranes as a petitio principii. It must, however, be pointed out that the same logical criticism applies to the diagnosis by similarity.

However, the use of the similarity method though faulty in logic is justified in fact. For though the diagnosis cannot be made a matter of certainty, it may be made with such a high degree of probability that there is no longer room for reasonable doubt. Some investigators have examined their twins with a plurality of refined methods which would be beyond the capacity of any single individual and only conveniently carried out by an anthropological institute. Such methods of reducing the chance of binovularity from one in millions to one in billions can only be necessary in exceptional cases. A case of mongolism in one only of monozygotic twins might well be examined by such elaborate methods, for instance : for so far anything approaching proof of such an event is lacking. For practical purposes, however, the diagnosis of uniovularity does not offer serious difficulties in the great majority of cases. A history of very striking similarity in appearance, supported by a short and convenient range of anthropological tests and measurements, including the very useful finger-print method, is adequate in the general run of cases. Nevertheless, it must be emphasized that the satisfactory diagnosis of ovularity is the necessary foundation of any reliable twin research, and its lack in the great majority of the earlier published cases very seriously detracts from their value. The method of diagnosis by membranes should not now be used unsupported by data from the similarity method.

At present there is little certain knowledge of the mechanism by which twins are produced. The commonly held opinion is that dissimilar twins are binovular and have arisen from the accidental production of two ova at the same time ; while similar twins are uniovular and have arisen from a single ovum by fission of the blastoderm, plural gastrulation, or by fission of the embryonic axis. In an interesting paper Curtius and von Verschuer (1932) investigated the frequency of twin-parenthood in the families of 931 twins. They came to the conclusion that there was a single recessive factor for twinning, which might

express itself in the homozygotic state as the parenthood of twins, was transmitted and exhibited equally by the male and the female, and showed no predilection for either the monozygotic or the dizygotic type of twin. The chance of this tendency to the production of twins manifesting itself at any particular birth they calculate (after Dahlberg) as 0·06. Their paper, as well as that of Meyer (1932), which goes in a similar direction, has been subjected to a destructive criticism by Lenz (1933), who considers that the capacity of twinning must be held to be a universal human characteristic and its manifestation governed by purely environmental factors. It would seem to the reviewer that the existence of a non-universal hereditary tendency to twinning has, however, not yet been disposed of, and that the theory of a recessive inheritance which may manifest itself in the homozygotic state in the actual twin pair, a possibility which is especially suggested by Meyer's paper, has not yet received adequate consideration. As a result of Lenz's criticism von Verschuer has subsequently (1933) retreated from his former position, but he still holds to the possibility of a uniovular origin of dizygotic twins, a rather far-fetched theory put forward by himself and Curtius to explain their finding that the father could be responsible for the production of dizygotic twins. The theory briefly is that the hereditary disposition to twinning manifests itself in a splitting tendency (Spaltungstendenz), which may be exhibited by the ovum or given to it by the sperm either after or before the final maturation division. They suppose that in the last of these cases, the ovum, before it has given up the second polar body, as a result of the Spaltungstendenz given it by the spermatozoon splits off a polar body which is as large and fertilizable as the remaining fertilized ovum, and that this polar body is itself fertilized by another sperm. The result is twins of differing constitution (e.g. perhaps differing sex) but uniovular origin. The possibility of such an event occurring had already been suggested by Oluf Thompson (1929). As Thompson stated, the result of this process would be two twins of identical maternal but differing paternal heredity, and the theory might be used to explain the occurrence of very similar opposite-sexed twins. Curtius' and von Verschuer's generalization would seem to be unjustified, however, as there is no evidence that such twins exist in any number, if at all. I cannot see that in their paper the authors recognize that this peculiar variety of twin would be the necessary result of the process they suppose to take place, or that they take note of the general opinion of embryologists that in man, as in animals, the division of the primary oocyte is the reduction division, that of the secondary oocyte is an equational division. The theory is fundamentally that put forward by Dahlberg, the "Spaltungstendenz" corresponding to Dahlberg's "doubling tendency." Neither theory provides any explanation for the increasing frequency of twinning with increasing age of the mother, and all the authors are constrained to suppose that polyovulation also occurs and is independent of any hereditary factor. The theory that there is a common hereditary disposition for the production of monozygotic and dizygotic twins, a theory to which von Verschuer appears to adhere, runs counter to the finding that in Japan there is a normal uniovular-twin birth-rate but a very low dizygotic-twin birth-rate.

It is commonly assumed that similar twins are identical in respect of their hereditary equipment. This cannot by any means be taken as proven. It is an admitted theoretical possibility that in the division of the fertilized ovum there might be an unequal division of the chromosomes. Such an event has been shown to occur in the reduction division in *Drosophila*, resulting in individuals with more or less than their proper quota of chromosomes, or in other cases with a fragment of a chromosome being additional to the normal quota in one individual, deficient in another. There is no cogent reason why this should not also occur in the somatic division, and in man. Furthermore somatic mutations are known to occur in a variety of different organisms, and if such a mutation were to occur in one of the two cells produced by the primary fission of the fertilized ovum it would produce a true hereditary difference in the two resulting monozygotic twins. These theoretical possibilities are, however, of little practical importance : for even were they shown to occur in man, they would be events of such rarity that statistical results obtained by the examination of large numbers of twins would be little affected by them.

A more important objection to the assumption of the identical hereditary constitution of monozygotic twins has been raised, among others, by Dahlberg (1926) and Newman (1923, 1937). These authors have emphasized the importance of asymmetrical phenomena occurring in monozygotic twins, and drawn attention to the frequency of " mirror-imaging," or of discordance in respect of such characters as handedness, unilateral nævi, asymmetrical characters of finger and palm-prints, etc. Both authors suppose there is an " asymmetry mechanism," specially affecting uniovular twins, which can be productive of real differences and cannot be called environmental in nature. Newman supposes that uniovular twins are derived from the partially developed right and left halves of a single embryo, and that the degree of asymmetry reversal depends on the lateness of the time of separation of the two halves to make new individuals. It seems, however, that this reversal of asymmetry occurs, especially as regards right- and left-handedness, in fraternal as well as identical twins. The figures provided by different workers are very conflicting. From Newman's compilation of his own figures and those of Dahlberg, von Verschuer, and Hirsch, given in his book (1937), and from the quotations from the literature and the original data supplied by Wilson and Jones (1932) the following table has been compiled :

	TOTAL NO. TWINS	PERCENT. LEFT		
		IDENTICAL	FRATERNAL	SINGLE BORN
In respect of : Handedness				
Newman's compilation (omitting ambidextrous)	1,018	16·3	7·9	—
Wilson and Jones	386	10·7	11·4	6·5
Crown Whorl				
Newman	190	24·4	4·1	—
Wilson and Jones	386	16·8	18·0	18·9
von Verschuer	386	25·6	26·3	—
Lauterbach	372	6·6 (like-sex)	18·1 (opp.-sex)	18·1
Dominant Eye				
Wilson and Jones	386	36·0	35·6	34·6

Von Verschuer (1932) provides a mass of data on the occurrence of asymmetries in human twins, and comes to the conclusion that the distribution of the asymmetrical character between the two twins is a chance phenomenon. If the reversal of the asymmetrical feature is due to the process that Newman hypothecates, then one would expect the presence of one asymmetrical feature to show a high correlation with the presence of others. Von Verschuer has worked out a series of these correlation coefficients, all of which proved not to be statistically significant. The two following tables are quoted from him:

	CORRELATION COEFFICIENT BETWEEN TWO ASYMMETRICAL FEATURES BY INDIVIDUALS	
	UNIOVULARS	BINOVULARS
Handedness and hand-folding	+0·16±0·12	+0·05±0·15
„ „ arm crossing	−0·18±0·12	−0·27±0·15
„ „ crown-whorl (direction).. ..	−0·18±0·12	+0·17±0·14
„ „ crown-whorl (site)	−0·13±0·15	−0·07±0·15
Hand-folding and crown-whorl (direction) ..	+0·16±0·16	+0·04±0·18

	CORRELATION COEFFICIENT BETWEEN CONCORDANCE (OR DISCORDANCE) OF TWO ASYMMETRICAL FEATURES, BY TWIN PAIRS	
	UNIOVULARS	BINOVULARS
Handedness and hand-folding	+0·08±0·12	+0·15±0·15
„ „ arm-crossing	−0·09±0·13	+0·03±0·12
„ „ crown-whorl (direction).. ..	−0·03±0·12	+0·07±0·14
„ „ crown-whorl (site)	0·00±0·13	−0·05±0·13
Hand-folding and crown-whorl (direction) ..	−0·08±0·16	+0·11±0·18

It is to be regretted that von Verschuer does not state the number of twins on which these figures are based. The absence of any correlation between any two asymmetries, however treated, does, however, provide a strong argument against Newman's hypothesis. Newman himself states that he has found correlation between various asymmetrical features, such as palm-prints and dental irregularities with handedness or crown-whorl, but his data are hardly given in sufficient detail to permit of any judgement.

The matter must therefore be regarded as still open. A judgement is made more difficult by the paucity of information on the frequency of these asymmetrical characters among the children of single births. So far it seems that left-handedness alone shows a higher incidence in twins than in singletons, and that in twins it is not yet certain that there is a higher incidence in the monozygotic than in the dizygotic. The increased frequency of left-handedness in fraternal twins as compared to the general population, *pace* Newman, does suggest that left-handedness in both varieties of twins may have a common explanation. This common explanation would be sought in the twin-pregnancy itself, a theory which would be quite in accordance with von Verschuer's failure

to find any correlation between the presence of one asymmetry and the presence of another. The matter, as Newman rightly points out, is not thereby disposed of. An individual in whom a partial reversal of asymmetry has occurred, e.g. an ambidextrous person, is not the same as one in whom it has not occurred, even where other qualities, such as psychological ones, are considered. If partial reversals of the functional asymmetry of the brain, from whatever cause, are of common occurrence in twins, they would be of fundamental importance for psychiatry. What their importance would be, however, would be a most difficult matter to assess. Whether this phenomenon of asymmetry reversal, if it occurs, be regarded as of environmental origin or not, there seems to be little sound evidence for Dahlberg's view that it can be accompanied by differences in the distribution of the germ-plasm. There is no more reason to suppose that two monozygotic twins, even showing discordance in asymmetrical features, are of different genotypic constitution than to suppose that the two sides of the body are different in this respect. As long as we are dealing with symmetrical characters or qualities expressed by the individual as a whole, as is mostly the case in psychiatry, we are justified in neglecting the theoretical possibility of inequalities in twin pairs from this cause.

So much for the theoretical basis of twin research. In a comprehensive review of its application in psychiatry in 1930 Luxenburger points out that work on twins may be classified into three kinds. There is the single-case report, the collection of a small or large number of cases in an unsystematic way, and the serial investigation in which all types of twins fulfilling certain conditions are examined, regardless of their ovularity, concordance, or discordance, etc. The great wealth of the literature is made up of work of the first kind, and, as Luxenburger has shown, it is for statistical purposes very largely useless. For it has been convincingly shown that when such reports of single cases are collected together and analysed they give no true picture of the twin material as provided by nature. They represent a selection of a particular type of case, the selection being in the first place for concordance. It is where *both* twins have suffered from schizophrenia, for instance, that the interest of the clinician has been aroused. These collections of single cases from the literature contain a quite undue proportion of concordant and of monozygotic twins, and even in the concordant cases there seems to be an undue selection for a far-reaching concordance down to the finest clinical details.

Luxenburger (1930*b*) provides a table compiled from the literature up to 1930, from which the following is extracted :

	UNIOVULAR		BINOVULAR		UNCERTAIN		TOTAL	
	CON.	DIS.	CON.	DIS.	CON.	DIS.	CON.	DIS.
Schizophrenia	31	3	3	5	9	2	43	10
Manic-depressive	24	1	1	1	2	2	27	4
Epilepsy	5	2	—	—	2	1	7	3
Psychopathic personality and hysteria	4	5	—	—	1	—	5	5
Oligophrenia	10	1	—	4	4	2	14	7
Mongolism	—	1 ?	—	22	7	16	7	39

Con. = concordant. Dis. = discordant.

It will be noted that, when mongols are omitted, over two-thirds of the reported twins are uniovular. The normal expectation would be to find about one-third uniovular. As exemplifying the differing value of the three types of work on twins, he provides the following striking table, showing the comparative results from (*a*) collection of cases from the literature ; (*b*) cases collected by circulars sent to doctors, institutions, etc. ; and (*c*) the systematic collection of an uninterrupted series :

FREQUENCY OF CONCORDANCE AND DISCORDANCE IN GROUPS OF TWINS GATHERED BY DIFFERENT METHODS, PER CENT.

	UNIOVULAR		BINOVULAR		UNCERTAIN		TOTAL	
	CON. %	DIS. %	CON. %	DIS. %	CON. %	DIS. %	CON. %	DIS. %
(*a*) Cases from literature	91	9	40	60	72	28	82	18
(*b*) Unsystematic collection ..	92	8	—	100	53	47	56	44
(*c*) Systematic collection	64	36	—	100	11	89	19	81

As Luxenburger says, the necessity for systematic unbiassed collections of twins may, on these data, be regarded as proven. Statistical analysis of the frequency of twinning in a clinical group, the extent of concordance in monozygotic twins as to clinical picture, onset, course, and outcome, is therefore impossible on the basis of such investigations. For the same reason uncontrolled collections, without guarantees against special and undesirable selection, are also unreliable. The last class of statistically unobjectionable serial investigations, securing a representative collection of twins, concordant and discordant, monozygotic and dizygotic, were in 1930 of great rarity ; and even to-day there are not many of them. It is largely due to Luxenburger that the merits of this type of investigation are now recognized and that psychiatrists have made as much use of the method as they have. The method, as he demands it should be carried out, is to start from a particular group of patients, selected on grounds of their being in particular hospitals at a particular time or otherwise having fixed and definite limitations of time and space, and by inquiries made at registry offices to discover all the twin births, irrespective of whether the twin survived or died, became subsequently ill or remained well, and to subject the whole of the material to an exhaustive examination. In this way a series is obtained which is without gaps and is representative : (1) in respect of the frequency of twins among the bearers of the character under investigation ; (2) in respect of the comparative frequency of like and opposite-sexed pairs, of monozygous and dizygous pairs, of concordance and discordance. Information on all these points may be of the greatest importance for any judgement to be reached on the presence, importance, and nature of hereditary factors. Even this type of investigation, as Luxenburger has said, is not without statistical source of error ; for by its means those twins are never discovered where, in spite of the presence of the hereditary factor, neither twin has developed the illness, abnormality, or character investigated. This source of

error is unavoidable, and must limit the value of all twin investigations. It is, however, often not feasible to approach Luxenburger's moderate ideal. It is in England, for instance, impossible to ascertain the fact of twinning from birth registers. Furthermore, for the final figures to be sound it would be necessary to make sure that the whole of the original material out of which the twins were selected is uniform—that, for instance, all possibilities of incorrect diagnosis have been eliminated. When a large twin material is being collected this is obviously not humanly possible. Most twin investigators will, therefore, have to content themselves with something less than what would be theoretically desirable. If the method of ascertainment of the twins is such that there is no likelihood of either uniovular or binovular, concordant or discordant twins being more largely represented in the final material than their proportional representation in nature, this is as much as can be demanded. The necessity of undiscriminating attention to all the material extends itself throughout the whole of the work, however, and it is necessary that the twins should not only be ascertained but also worked up with equal care, regardless of whether they are monozygotic or dizygotic, concordant or discordant. Furthermore, the reliability of any such investigation demands the satisfactory diagnosis of ovularity.

This is not to assert that all case reports of single cases are without value. A particular character may be of such rarity that its occurrence in a pair of twins is an event of importance and worthy of record. In such cases clinicians should be aware of the fact that all twins, monozygotic or dizygotic, concordant or discordant, are of importance. It is, however, obviously better that such twins should be ascertained by an automatic method which does not depend for its certainty on the interest of the clinician. All hospitals where any research work is done at all should make inquiries as to twinship about all their patients as a matter of ordinary routine, so that when an interested investigator appears he may have a chance of following up *all* cases showing a particular syndrome.

Twin Research in Mental Defect

In attempting a summary of the results obtained by twin investigations in psychiatry, and taking at first the subject of mental defect, the large number of case reports that have been made on cases of mongolism occurring in twins provide an elegant example of the weakness of the methods used hitherto. In spite of the rarity of the condition, it has aroused so much clinical interest that Rosanoff in 1934 (1934*a*) was able to collect 59 cases from the literature, and his collection was by no means complete. To these 59 cases he was able to add 5 new pairs of his own. Of these 64 pairs, in five the sex of the twins is not stated. It is difficult to imagine why a clinician can have left out such an important point in his report. A further 23 pairs were of opposite sex. This is about the number one would expect in a random selection ; so mongolism forms the only exception to the rule stated by Luxenburger to the effect that cases from the literature represent a selection for uniovularity. Rosanoff states that

eight pairs were monozygotic and all concordant. All the same-sexed pairs held to be dizygotic and all the opposite-sexed pairs were discordant in respect of mongolism. These findings might by themselves be interpreted as indicating that a hereditary factor played a major rôle in the ætiology. Rosanoff, however, agrees with the majority of other workers in thinking that the condition is not hereditary, and that the twin findings do not stand in contradiction to this belief. Family investigations do not disclose an increased frequency of mongolism among the sibs of mongols. The most widely held theory of the causation of mongolism is that the condition is due to faulty implantation of the ovum in an abnormal uterine mucosa. Monozygotic twins with closely neighbouring implantation sites and with a common placenta would naturally be likely to be concordant in regard to a character caused by deficient or abnormal nutrition consequent on the location of the placenta. Rosanoff's theory is that the condition is caused by injury to the ovum on ovulation, the injury being brought about by scars produced in the ovary by previous ovulations. The frequency of mongolism would on that theory naturally increase with increasing age of the mother, as has repeatedly been shown to be the case. One of the cases which Rosanoff has not collected from the literature is Siegert's case (1922, quoted by Siemens 1924) of discordant monozygotic twins. Were the uniovularity of Siegert's twins established (no evidence beyond a statement is provided), it would be a direct proof of the incorrectness of Rosanoff's theory, which obviously demands that all monozygotic twins without exception should be concordant.

The fact is that no definite statement can be made about the incidence of mongolism in monozygotic and dizygotic twins from cases collected from the literature, as Rosanoff has collected them. The diagnosis of ovularity is not satisfactorily carried out in one of them. Nevertheless, the example of mongolism provides a lesson in the necessity of caution in the interpretation of concordance-discordance differences in monozygotic and dizygotic twins as a direct measure of the relative prepotence of heredity. In any case where a particular character may be the result of intra-uterine processes or of the process of twinning, it is clear that the results of researches into twins must be interpreted with especial caution.

Apart from mongolism, case reports on the occurrence of special forms of mental defect in twins are few and unimportant. Dorff (1934) has reported a case of cretinism occurring in one of binovular twins; in uniovular twins Wilson and Wolfsohn (1929) have reported cerebral deplegia with mental defect and congenital nuclear ophthalmoplegia with mental defect, von Engerth (1933) tower skull, Fabing (1934) epiloia, Jenkins and Glickman (1934) birth injury (one only affected). In most cases a selection of the appropriate literature is given. In Fabing's case, which appears to be unique, no data are given relevant to the diagnosis of ovularity. In both his and von Engerth's case there are interesting data regarding mirror-imaging in pathological features. Von Engerth's case is beautifully worked up from the biometric aspect. A not uninteresting case is reported by Schmitz-Lückger (1932), who observed mental defect of about the same degree and with many clinical similarities in a pair of

opposite-sexed twins, and suggests it was due to the contraceptive employed by the parents, which had not prevented the occurrence, but had damaged the result of conception.

Of the more usual types of mental defect a number of single cases occurring in twins have been reported, which it is not worth while to detail, as more valuable material is available. Both Smith (1929, 1930) and Rosanoff (1931, 1934) have made fairly systematic investigations in this field.

Rosanoff (1931) has published his results with mentally defective twins with a brevity that robs his paper of much value. He gives no account of the methods of selection, no account of the cases from a clinical point of view, no account of the methods used for the diagnosis of ovularity, nor is there any statement of any quantitative measurement of the degree of defect. The body of the paper is given over to a discussion of a theory of the method of inheritance which will be dealt with later. From the brief and incomplete analysis he gives of his material the following table can be constructed:

	CONCORDANT	DISCORDANT	TOTAL
Monozygotic	33	2	35 pairs
Dizygotic, same-sexed	21	12	33 „
Dizygotic, opposite-sexed	11	16	27 „

Of the 16 discordant opposite-sexed pairs, the male was affected in 11 cases, the female in five. In all there were 91 mentally defective in 101 males, 69 mentally defective in 89 females. Rosanoff attempts to interpret his findings as reconcilable with a dimeric inheritance of mental defect, the factors being in both cases recessive, one autosomal, the other located in the X-chromosome. He finds support for this theory in the results of the family investigations of Goddard, which he has subjected to an analysis with this theory in view. In a later paper (1934) he reports the findings in a further 65 (making in all 81) pairs of opposite-sexed twins. Among these he found 34 pairs where both twins were mentally defective, 31 where the male alone was affected, 16 where the female alone was affected. He further reports the findings in 233 pairs of opposite-sexed twins, of normal or superior intelligence (I.Q. 76–161), and found that the girls tended to have a higher intelligence than the boys. He further examined the intelligence scores of the parents of 100 of these twins and found that the fathers had higher I.Qs. than the mothers in 57 of the 100 cases. The sex-distribution of higher I.Q. in the parents had no correlation with the sex-distribution of higher I.Q. among the twins—a fact that speaks strongly against Rosanoff's theory, though he does not treat it as such. On this basis Rosanoff and his co-workers build up a theory of the greater vulnerability of the male fœtus and sex-linked recessive inheritance as independent causes of the higher intelligence among the girls. A full analysis of Rosanoff's figures and his argument would not repay itself, but there is one important possibility to be pointed out. Rosanoff nowhere states what the ages of his twins were. It

seems extremely probable that he was dealing with children, as he speaks everywhere of boys and girls. Now it is a well-known phenomenon that in the curves of growth of intelligence with age of boys and girls, the curve is steeper in the case of girls than boys, and that in consequence the girls reach maturity in intelligence before the boys do. If this factor has not been taken into account by Rosanoff it would make just such a systematic difference between male and female twins as he observed.

Luxenburger, stimulated by Rosanoff's theory, has published (1932) an analysis of the material of Brugger and Lokay, which as it is not a twin-material, is not the immediate concern of this review. Nevertheless, the figures he supplies are of interest and importance. In the families of 209 propositi he found the frequency of mental defect among children classified according to the constitution of their parents to be as follows :

	SONS	DAUGHTERS
Both parents normal	0·212 ± 0·085	0·097 ± 0·020
Father defective, mother normal	0·404 ± 0·068	0·577 ± 0·097
Father normal, mother defective	0·538 ± 0·098	0·423 ± 0·068
Both parents defective	0·925 ± 0·041	0·892 ± 0·051

Luxbenburger analyses the expectations of what these proportions would be on the theories (1) of simple autosomal recessive inheritance, (2) of simple recessive sex-linked inheritance, (3) of both methods of inheritance occurring in a mixed population, (4) of dimeric recessive autosomal and sex-linked inheritance (Rosanoff). He finds that the last theory provides the best explanation of the facts. Luxenburger's mathematics are not above reproach. He has, for instance, made no allowance for the comparative frequencies of the different types of crossings which would be productive of defectives. The mathematics of this problem are certainly more complicated than Luxenburger represents them, and would be worth the attention of a mathematical specialist. I think it would be found that Rosanoff's theory provides an inadequate explanation of the empirically found figures, and it seems to me likely that it would involve a greater relative frequency of mental defect in males as compared with females than is actually found. Rosanoff is himself aware of the inadequacy of his theory, and brings in the supplementary hypothesis of the greater vulnerability of the male fœtus. Luxenburger also does not suppose that this method of dimeric inheritance would be applicable to all cases of hereditary mental defect, but thinks that other methods of inheritance also play a part, if only a minor one. The chief criticism of the work of these two authors must be that it represents an over-simplification of the problem. Human beings are not simply to be divided into normal and defective. Any theory of the inheritance of mental defect must provide an explanation of the curve of normal distribution of intelligence found in both males and females. It seems to me that the only two theories that will simply explain this phenomenon are : (1) that there are

a large number of factors for mental defect, which are of different frequency in the population, and each produce mental defect of different degrees ; or (2) that there are a large number of factors for intelligence, any number of which may be present in a single individual, the effect of any one of which will be to enhance the action of any of the others—that is, the more of these factors anyone has, the more intelligent he will be, the less the less. The two theories are not mutually exclusive and there is much to suggest that both have some truth. But even that represents an over-simplification, as among other things the environment must play a rôle.

The effect of the environment on intelligence and character has been the subject of an elegant and painstaking investigation of Newman, Freeman, and Holzinger (1937), an investigation which will probably remain unique. It must be briefly referred to, as it is not principally concerned with mental defect. The unique part of the work consists in the collection of 19 pairs of monozygotic twins who had been separated in early life and brought up in different environments. The authors were able to show that the intelligence, as estimated by standard tests, is subject to greater variation with varying environment than is commonly supposed. The same was true of character-traits, as estimated by other tests ; but this part of their work is less satisfactory, owing to the greater difficulty in interpreting the meaning of such tests. The result of their work is to throw into relief the limitations of twin work and to illuminate the fundamental difficulty of stating what the problem is, which is involved in the often formulated antithesis, heredity *vs.* environment. It is clear that if one were to take two individuals of like innate ability, and subject one from infancy to adult life to a stimulating and well-balanced education, and to keep the other from education of any sort whatever, the final result would be very different in the two cases. This is what the above-named authors have shown, in a less extreme case, to be true. Under the widest range of environmental circumstance they found the extreme range of variation in intelligence quotient to be 24 per cent. In this case the variation was from 8 points below to 16 points above the normal value of 100 per cent. It is not admissible to argue from this that at lower or higher levels of intelligence a comparable change in the environment would produce an equal change in the test-intelligence. Both environment and heredity in the individual set limits to the range of variation in intelligence, of which the lower limit is nil. If the environment is depressing, an improvement in it may be expected to produce a marked improvement in the intelligence. If, however, it is already favourable, a comparable variability cannot be expected. That quite large changes in the environment do not as a rule produce large changes in the I.Q. is shown by Holzinger's calculations that the mean difference in I.Q. in these 19 twins was little more than 8 points. That it was found to be as much as this will be a matter for surprise for some.

The investigation of Newman, Freeman, and Holzinger was principally concerned with the effect of the post-natal environment. To the prenatal environment and to the various environmental influences brought to bear at the time of birth, a probably much larger rôle is to be ascribed. The effect of

birth-trauma in producing mental defect has not yet been satisfactorily investigated. Brander (1935) has drawn attention to this, and has criticized especially the work of Smith, still to be discussed, as taking insufficient account of birth-trauma as a cause of defect, especially in concordant twins. In his own series of 10 mentally defective twin-pairs birth anomalies had played a considerable rôle, and the twin with the lower I.Q. had in every case been subjected to their influence. He emphasized especially the importance of minor degrees of birth-trauma in producing the less gross degrees of mental retardation. There seems to be much justice in his criticism ; and there is no reason to think that birth trauma, like the environmental factors responsible for mongolism, may not produce mental defect in both of a pair of uniovular twins.

Smith gives an account of his investigations in two papers. An account at length is given in the Danish language (1929), and is not available to the reviewer. A shortened account is also given in German (1930), and appears to contain the more essential facts. In the course of the registration of all the mental defectives in Denmark, Smith ascertained all the multiple births among the first 6,700 mental defectives registered. In 67 pairs of twins full details were available. Of these 35 pairs were of opposite sex. A careful attempt was made to establish the ovularity of all the same-sexed pairs, and though this was not possible with certainty in every case (for instance where the twin partner was already dead), there is no reason to think that errors of ovularity diagnosis are a large source of error in his results. He found in all 50 pairs of binovular twins, of whom four were concordant in respect of mental deficiency. In one case no decision could be reached as to ovularity. In three cases uniovularity was regarded as probable : all of these were concordant. Thirteen pairs were regarded as certainly uniovular, of whom 11 pairs were concordant, two discordant. The proportion of concordance in uniovular pairs (11/13 =84·6 per cent.) compared to that in binovular pairs (8 per cent.) speaks, as Smith remarks, strongly for the hereditary determination of the majority of cases of mental defect. Smith's cases were all quite ordinary cases of mental defect, with or without history of fits, with or without neurological abnormalities. There were no cases of the rarer and special forms of mental defect. He was unable to find a clinical picture that was characteristic for a hereditary or for an acquired form of the condition. His most interesting finding is that a number of the concordant uniovular pairs showed various neurological abnormalities. In pair 109 both twins had a left-sided spastic hemiplegia, both had convulsions as children ; one of the pair got progressively worse and died at the age of 22 ; the other improved, though neurological signs remained. Both twins of pair 115 showed disturbance of speech, with defective articulation, both were somewhat deaf, one showed a positive Babinski. In another three pairs (perhaps four) both twins had convulsions, and one of one pair died in status epilepticus. This conformity of neurological picture Smith is inclined to interpret as suggesting that even those cases of mental defect which show an abnormal neurological picture may be due to hereditary and not to any exogenous cause. The possibility must be conceded, but the evidence is

not conclusive. Other explanations cannot be excluded, as in the case of mongolism.

Smith's two discordant uniovular pairs both show an obviously exogenous disorder. In pair 120 the affected twin had fits from the third day after birth, recurring at intervals since. The left side of the body became with further growth markedly underdeveloped, and he died in a state of idiocy at eight; his brother was normal. This would appear to be a case of birth trauma. In case 121 (triplets), the affected twin and his sister had a severe exanthematous illness at the age of three months, by which the healthy brother remained unaffected.

As regards the degree of defect, Smith does not provide very detailed information in his German paper ; but from what facts he does give it is clear that there were perceptible though not very large differences in this respect Only in one pair (116) is the difference at all a marked one. Characterological differences were also noted in the concordant twins. One gets the impression from the work that an inborn tendency to mental defect manifests itself in a high proportion of the cases where it is present, and generally to its full effect. In other words, the gene or genes responsible show, in the language of genetics, high values for manifestation rate, expressivity, and specificity. This is not to say that environmental factors are not important. There may be cases of mental defect largely determined by heredity where the environment is largely without power of modification, and others caused solely by the environment (e.g birth trauma) where there has been no hereditary predisposition.

Working on Smith's material, Luxenburger has attempted to obtain an actual value for the manifestation rate of the hereditary factors responsible for mental defect. By manifestation rate is meant the average percentage of cases where the genotypic factors are present in which they show themselves phenotypically.

Luxenburger provides a formula

$$\mathrm{M}=\frac{m_g-pvy'}{(z-2p)y'}$$

in which M represents the manifestation rate, m_g the number of concordant twins among the like-sexed pairs, v the empirically found expectation of sibs to be affected similarly to the propositus (in this case with mental defect), y' the expectation of a twin reaching the average age of the material investigated and z the total number of twin proposity. This formula is for use on a total twin material, including all cases that died in infancy, and can only be applied to material gathered by a method of selection where such factors as age, sex concordance, etc., have no selective effect. Its great convenience is that it avoids the determination of ovularity altogether. Its weakness lies in the fact that the factors v and y' are themselves subject to large errors. From Smith's figures Luxenburger calculates the " expected figure " for the manifestation rate of the genes of mental defect as 0·835, and compares this with the " observed figure of 0·846 (the number of concordant uniovular twins divided by the total number of uniovular twins, here 11/13). Luxenburger points out that the agreement is good. It is, however, without significance.

The " manifestation rate " of an inherited disposition is a term derived from animal genetics. It signifies that where the inherited tendency towards the development of a certain character is present it will manifest itself in a certain proportion of cases. In the remainder it will remain inhibited. The inhibition may be either by the action of other inherited factors or by the presence of an unsuitable environment. If the inhibition is solely by reason of other inherited factors, it will take place, if at all, in both of a pair of twins, which will then entirely escape observation. The calculation of the manifestation rate from concordance-discordance figures from twin studies gives only an expression for the effect of the environment. The environment may, however, be of such a quality that the character is produced (or imitated) without the inherited tendency being present at all. This is probably the case with Smith's two discordant pairs, where there are certainly no grounds for the assumption that there was a hereditary disposition to mental defect present in both twins which showed itself in only one. These two cases would more properly have been excluded from the calculation, and the " observed " manifestation rate is in Smith's cases apparently 1·0.

Luxenburger, however, in his analysis of Smith's results is concerned with a particular point. He quotes the results of Brugger and Lokay, who investigated the frequency of mental defect among the sibs of defectives, and found it to be 0·13 to 0·18. Both figures are well behind the expected 0·25 on the assumption of simple recessive inheritance. Luxenburger is concerned to show that this discrepancy is not due to deficient manifestation of the inherited factor. It is clear that a manifestation rate of 0·835 or 0·846 or 1·0 will not explain the total discrepancy, but at most a part of it. Luxenburger seems not to have been aware at this time of the possible additional disturbing effect of other inherited factors than the specific ones (factors conveniently classed as the " genotypic milieu "), but only to have reckoned with possible environmental interference. As Luxenburger has himself subsequently pointed out, the effect of the genotypic milieu is most conveniently to be measured together with the effect of the environment (when certain assumptions are made as to the mode of inheritance) by discovering the frequency of a character among the children of parents, both of which show it. From Luxenburgers figures, previously quoted, it appears that the incidence of mental defect among the children of two defective parents is about 0·9.

Luxenburger next considers the possible influence of lethal effects of the genes concerned. Obviously if a particular gene were to have a lethal effect the number of its carriers in a given population would be reduced and in families known to carry the gene the number of those giving evidence of its presence would be below the expected value. Even if the lethal effect were only exhibited in a percentage of cases, this would still be the case. Obviously the genes for mental defect are not absolutely lethal, because then there would be no subjects of mental defect that would survive. Luxenburger attempts to test the possibility of a lethal effect showing itself in a proportion of cases by calculating the relative and absolute frequency of uniovular and binovular pairs in Smith's material.

The method is based on a statement of Weinberg's to the effect that among the bearers of a character which involves an increased prenatal mortality, a relatively reduced percentage of binovular twins is to be expected. Luxenburger calculates that the percentage of twins in Smith's material (122 in 6,689) corresponds to average expectation and that the opposite-sexed twins (i.e. certainly dizygotic twins) are neither more nor less frequent than could be expected. He deduces from this that the fact of twinning as such does not predispose to mental defect, and that there is no evidence of any lethal effect exercised by the genes of mental defect.

These conclusions may be true, but the reasoning does not seem to be altogether sound, and there are reasons to be sceptical. Twin conception is known to predispose to increased prenatal mortality, twin birth to birth injury, and birth injury to mental defect and to reduced viability. Luxenburger seems to have made no direct comparison of the duration of life of the twin partners in Smith's material with the life tables of the general Danish population, a population of defectives, or with a population of normal twins. Facts gained from such comparisons would be more valuable than the somewhat indirect evidence supplied.

Luxenburger's argument, that the normal distribution of uniovular and binovular twins in Smith's material is against the assumption of any lethal effect, rests entirely on the validity of Weinberg's statement that a lethal effect would show itself in a reduced number of binovular twins. Luxenburger does not give a reference to the particular work of Weinberg's in which this statement occurs and I am unable to trace it. The statement itself, though proceeding from a statistical authority, seems to me of questionable truth.

One might approach the problem as follows :

Taking a group of crossings which produce the character under discussion, let :

a = the number of uniovular twin pairs born,
b = the number of binovular twin pairs born,
v = the frequency of the character in the children of these crossings.

Then there will be :

av uniovular pairs with the character,
$a(1-v)$ uniovular pairs without the character,
bv^2 binovular concordant pairs,
$2bv(1-v)$ binovular discordant pairs,
$b(1-v)^2$ binovular pairs without the character.

Starting from twin propositi one will discover (by Weinberg's " Probandenmethode," used in all the German calculations) :

$2av$ concordant uniovular pairs,
$2bv^2$ concordant binovular pairs,
$2bv(1-v)$ discordant binovular pairs.

The relation of the uniovular twin pairs found to the binovular pairs found will be :

$$\frac{2av}{2bv^2+2bv(1-v)}=\frac{a}{b}.$$

Now let the character have a lethal effect e which is exerted at random. Among the uniovular pairs who bear the character there now will be :

$av(1-e)^2$ pairs where both twins survive,
$2ave(1-e)$ pairs where one twin survives,
ave^2 pairs where neither twin survives.

Among the binovular twins there will be :

$bv^2(1-e)^2$ concordant pairs where both twins survive,
$2bv^2e(1-e)$ concordant pairs where one twin survives,
bv^2e^2 concordant pairs where neither twin survives,
$2bv(1-v)(1-e)$ discordant pairs where both twins survive,
$2bv(1-v)e$ discordant pairs where one twin survives.

Starting again from twin propositi one now will find :

$2av(1-e)^2$ uniovular twin pairs, all concordant,
$2bv^2(1-e)^2$ binovular twin pairs, concordant,
$2bv(1-v)(1-e)$ binovular pairs, discordant.

and the relation of uniovular to binovular twin pairs found will be

$$\frac{2av(1-e)^2}{2bv^2(1-e)^2+2bv(1-v)(1-e)} = \frac{a}{b} \cdot \frac{1-e}{1-ve}.$$

But as v and e are by hypothesis both positive fractions, $\frac{1-e}{1-ve}$ must be less than 1. This resolves itself to a statement that under the conditions laid down above, the lethal effect of a hereditary factor would show itself in a *larger* and not a smaller proportion of dizygotic twins in a total twin material.

The fundamental assumption in the above argument is that the lethal effect is exhibited in a proportion of cases distributed at random. If the lethal effect were dependent for its manifestation on favourable environmental circumstances, this might well be the case. If, however, the lethal effect were dependent on a favourable genotypic milieu, the result would be different.

Let us suppose that the character exhibits its lethal effect only where are a considerable number of certain other common genes also present. These other genes are only found all together in a proportion e of cases. Now the circumstances among the binovular twins will remain as in the example worked out above ; but among the uniovular twins the lethal affect will show itself in a proportion e of pairs, in all of which both members of the pair will be affected. Starting again from twin propositi, the number of monozygotic twin pairs found will now be no longer $2av(1-e)^2$, but $2av(1-e)$; and the relation of uniovular to binovular twins found will be no longer $\frac{a}{b} \cdot \frac{1-e}{1-ve}$ but $\frac{a}{b} \cdot \frac{1}{1-ve}$. In this case the proportion of dizygotic twins found will be unduly low, as Weinberg is said to have stated.

But we do not know whether this purely hypothetical lethal effect is exhibited in accordance with one or other or both of the possibilities outlined above. It is therefore not admissible to argue from a normal distribution of uniovular and binovular twins that no lethal effect of the genes concerned is present. The

matter is not of purely theoretical interest, as it might appear, for in the twin work on epilepsy we shall meet with it again and find far-reaching conclusions based on arguments similar to Luxenburger's.

If we attempt a summary of what twin work has added to our knowledge of the inheritance of mental defect we may say that its findings are in conformity with those supplied by family investigations, and indicate that hereditary factors are of great importance, though certainly not the only ones concerned in the production of the syndrome. Much further investigation is required, and not least in the rarer and special forms of mental defect. The systematic ascertainment of all twins in institutions for mental defectives would be likely to be productive of a very valuable material for research.

REFERENCES

Brander, T. (1935). *Mschr. Kinderheilk.*, **63,** 276.
Conrad, K. (1937). *Fortschr. Neurol. Psychiat.*, **9,** 197.
Curtius, F. (1930). *Arch. Gynäk.*, **140,** 361.
Curtius, F., and Verschuer, O. v. (1932). *Arch. Rass. u. GesBiol.*, **26,** 361.
Dahlberg, G. (1926). " Twin Births and Twins from a Hereditary Point of View." Stockholm.
Dorff, G. B. (1934). *Amer. J. Dis. Child.*, **48,** 1,316.
Engerth, G. v. (1933). *Z. ges. Neurol. Psychiat.*, **148,** 670.
Fabing, H. (1934). *Brain*, **57,** 227.
Jenkins, R. L., and Glickman, E. (1934). *Amer. J. Dis. Child.*, **48,** 130.
Lange, J. (1929). *Allg. Z. Psychiat.*, **90,** 122.
Lassen, M. T. (1931). *Arch. Gynäk.*, **147,** 48.
Lenz, F. (1933). *Arch. Rass. u. GesBiol.*, **27,** 294 and 311.
Luxenburger, H. (1929). *Fortschr. Neurol. Psychiat.*, **1,** 82.
—— (1930*a*). *Ibid.*, **2,** 373.
—— (1930*b*). *Zbl. ges. Neurol. Psychiat.*, **56,** 145.
—— (1931). *Z. ges. Neurol. Psychiat.*, **135,** 767.
—— (1932*a*). *Fortschr. Neurol. Psychiat.*, **4,** 1 and 49.
—— (1932*b*). *Z. ges. Neurol. Psychiat.*, **140,** 320.
—— (1932*c*). *Z. indukt. Abstamm. u. VererbLehre*, **61,** 223.
—— (1933). *Fortschr. Neurol. Psychiat.*, **5,** 1.
—— (1935). *Ibid.*, **7,** 173.
Meyer, H. C. (1932). *Arch. Rass. u. GesBiol.*, **26,** 387.
Newman, H. H. (1923). " The Physiology of Twinning." Univ. Press, Chicago.
Newman, H. H., Freeman, F. N., and Holzinger, K. J. (1937). " Twins : a Study of Heredity and Environment." Univ. Press, Chicago.
Rosanoff, A. J. (1931). *Amer. J. Psychiat.*, **11,** 289.
Rosanoff, A. J., and Handy, L. M. (1934). *Amer. J. Dis. Child.*, **48,** 764.
Rosanoff, A. J., Handy, L. M., Rosanoff, I. A., and Inman-Kane, C. V. (1934). *J. Nerv. Ment. Dis.*, **80,** 125.
Schmitz-Lückger, J. (1932). *Psychiat. neurol. Wschr.*, **34,** 437.
Siegert, . . (1922). Contribution to discussion in *Klin. Wschr.*, **1,** 2,259.
Siemens, H. W. (1924). " Die Zwillingspathologie." Springer, Berlin.
—— (1932). *Z. indukt. Abstamm. u. VererbLehre*, **61,** 208.
Smith, J. C. (1929). *Medd. Danm. Antrop.*, **3,** 1 (quoted Smith 1930, Luxenburger 1931, q.v. —not verified).
—— (1930). *Z. ges. Neurol. Psychiat.*, **125,** 678.
—— (1931). *Acta Psychiat., Kbh.*, **6,** 79.

Thompson, O. (1929). Quoted Smith (1931), q.v.
Verschuer, O. v. (1925). *Münch. med. Wschr.*, **72**, 184.
—— (1927). *Ergebn. inn. Med. Kinderheilk.*, **31**, 35.
—— (1932). *Z. indukt. Abstamm. u. VererbLehre*, **61**, 147.
—— (1933). *Arch. Gynäk.*, **156**, 362.
Wilson, S. A. K., and Wolfsohn, J. M. (1929). *Arch. Neurol. Psychiat.*, **21**, 477.
Wilson, P. T., and Jones, H. E. (1932). *Genetics*, **17**, 560.

EPITOME OF CURRENT JOURNALS

ALLGEMEINE ZEITSCHRIFT FÜR PSYCHIATRIE UND IHRE GRENZGEBIETE

Vol. 107. Nos. 1 and 2. March 1938.

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Insulin and Cardiazol Treatment of Schizophrenia.—Critical review of the literature.

Treatment of Schizophrenia.—The complications which occur during insulin treatment may be divided into three groups: (1) disturbance in organs other than the central nervous system (circulatory, respiratory); (2) disturbance of absorption of sugar given during the shock; (3) deep coma, which may last for several days, and may be "akinetic" or "hyperkynetic" (with extrapyramidal symptoms or epileptic seizures). Suggestions to prevent or counteract these complications are given. (K. S.)

Insulin Shock Treatment of Schizophrenia.—Symptoms of restlessness, excitement, and hallucinations could be influenced to an extent which was never observed after other forms of somatic or psychological treatment. (K. S.)

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Children receiving Benzedrine Sulphate.—Twenty-one boys and nine girls, aged 5–14 years, who were already resident in the hospital suffering from a variety of behaviour disturbances, were selected for study. Every child in the group had been under observation for at least a month. A daily dose of benzedrine was administered on waking. Fourteen children exhibited a marked improvement in their school work, both as regards interest displayed and industry. Eight did not show such a striking change, and five showed no change in school performance, though their behaviour in other respects was distinctly altered. Two children showed no response to the drug at all. Nearly all the children became emotionally subdued, without, however, losing interest in their surroundings. All the children were given a trial dose of 20 mg., but in eight children this dose was reduced to 10 mg. following gastro-intestinal upsets. One child did not react at all to this dose.

Mental Changes following Removal of Brain Tumour.—A woman aged 38 suffered from severe headaches for one and a half years. Examination showed only a very slight weakness of the left lower face and mild euphoria. X-rays demonstrated extensive calcification in the right frontal region. The partial removal of a right frontal astrocytoma was performed. Symptoms of increased intracranial pressure appeared and mild mental changes appeared about six months later. One year after the first operation a second more radical removal of the tumour was carried out. Ten months later, on account of headaches and mental changes, a third operation was performed and most of the right cerebral hemisphere was removed. When examined six months after the operation the patient showed some emotional instability, slight impairment of recent memory, but little if any deterioration in her intellectual ability as measured by mental tests. Eight months after the operation, the strength in the left leg had improved sufficiently to enable her to walk. She could raise her left arm about six inches from the lap, but apparently could perform no other voluntary movement in the upper extremity. The cranial nerves were entirely normal with the exception of a left homonymous hemianopia.

Defects of Intelligence from Focal Lesions.—Clinical and anatomical records of seven cases of right hemiplegia are presented and a study of the intellectual defect indicates that this is an affection or group of related affections more or less separable from such associated disturbances as aphasia, apraxia, paralysis, sensory loss, and emotional disorder. A review of the correlated clinical and pathological studies of these cases tends to localize the structures from which intellectual defect is produced by injury. Both negative and positive evidence tends to indicate that these structures occupy a relatively small area situated posteriorly in the basal region of the brain.

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Cortical Atrophy in Presenile Psychoses.—The author gives a detailed description of two cases. The first autopsy revealed marked atrophy of the temporal convolutions, hippocampus, and part of the frontal convolutions of the left side. There was distortion in the arrangement of the cyto-architectural layers of the left temporal lobe, and retrograde changes in the ganglion cells. There were also marked swellings of the nerve cells near the paraolivaris dorsalis. The atherosclerotic changes of the smaller cortical vessels were considered as a possible cause of the atrophy. The diencephalic region, which showed similar atherosclerotic changes of the vessels, did not contain any alterations of the ganglion cells. The absence of senile plaques and of Alzheimer's fibrillary nerve cells favoured a diagnosis of Pick's disease. In the second case examined there was a convolutional atrophy of the left hemisphere involving chiefly the left first frontal, first and second temporal convolutions, and Ammon's horn (Sommer's sector), characterized by chromatolysis of nerve cells and increase in glia cells. Against a diagnosis of Alzheimer was the absence of fibrillary changes in the nerve cells and senile plaques. The various pathogenetic theories accounting for the production of the disorder are discussed.

Protracted Hypoglycæmia.—A case of a protracted coma lasting three days in a 29-year-old schizophrenic girl is described. Sucrose by the mouth and intravenous glucose were duly administered. The clinical picture did not change in spite of the fact that the blood sugar level varied from 30 mg. to 235 mg. It is suggested that the nerve cells were so injured by the hypoglycæmia that they were rendered incapable of utilizing the carbohydrates that were available, and required a period of 49 hours to recover. The outcome was favourable. The improvement noted in the psychosis began with her recovery from the protracted coma and has continued up to the present, when the patient is again able to enjoy the mental health she had prior to the onset of her illness.

Vitamin C Studies in Alcoholics.—Vitamin C studies on the blood and spinal fluid as well as the five-hourly excretion test of Wright were carried out on a large number of normal patients and a group of alcoholic patients in the psychiatric and neurological wards of Bellevue Hospital. The alcoholic cases were clinically divisible into ten groups—

Group I: Chronic alcoholism without psychosis or peripheral neuritis (nine cases). These vitamin C values fall within normal range.

Group II: Chronic alcoholism with peripheral neuritis (eight cases). The urinary excretion test was done on two cases and yielded low figures. No observations were made on the blood or C.S.F.

Group III: Alcoholic hallucinosis with mild peripheral neuritis (10 cases). The figures for blood and C.S.F. were far below normal in nine cases, and normal in one case.

Group IV: Alcoholic hallucinosis (35 cases). In all cases the Vitamin C levels were below normal in blood and C.S.F.

Group V: Alcoholic hallucinosis—observations on cases immediately after recovery (six cases). The average blood and C.S.F. values were higher than in the preceding group, but still below normal.

Group VI: Delirium tremens (14 cases). All vitamin C values in blood and C.S.F. were below normal.

Group VII: Korsakoff psychosis (five cases). In only one case was a normal figure obtained for C.S.F., and here the blood was low. All other cases gave subnormal values.

Group VIII: Alcoholic encephalopathy (five cases). In every case vitamin C was below normal in blood and C.S.F.

These results emphasize the nutritional factor in the production of nervous and mental changes in chronic alcoholics.

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Intro-Extraversion: the Concepts and their Clinical Use. R. M. Collier and M. Emch. 1,045.
The Appraisal of Intellectual and Physical Factors after Cerebral Damage in Children. B. Crothers and E. Lord. 1,077.
The Other Side of Hallucinations. J. Lang. 1,089.
A Review of the Results from the Employment of Malaria Therapy in the Treatment of Neuro-Syphilis in the Florida State Hospital. M. F. Boyd, W. K. Stratman-Thomas, S. F. Kitchen, and W. H. Kupper. 1,099.
Hereditary Form of Primary Parenchymatous Atrophy of the Cerebellar Cortex associated with Mental Deterioration. A. J. Akelaitis. 1,115.
A Constructive Criticism of Certain Hospital Procedures. K. H. Bowman. 1,141.
Statistical Contributions from the Mental Hygiene Study of the Eastern Health District of Baltimore. B. M. Cohen and R. E. Fairbank. 1,153.
*The Diagnostic Validity of the Rorschach Test. J. D. Benjamin and F. G. Ebaugh. 1,163.
*Some Clinical and Physiological Aspects of the Brain Potentials. J. Hughes, E. A. Strecker, and K. E. Appel. 1,179.
Post-Operative Psychoses. M. M. Abeles. 1,187.
*Results of 15 Years' Experience with the Ketogenic Diet in the Treatment of Epilepsy in Children. H. F. Helmholz and M. Goldstein. 1,205.
Some Data of the Psychiatric Morbidity of Jews and Arabs in Palestine. L. Halpern. 1,215.
Psychiatric History of the Development in California. M. H. Smyth. 1,223.

Diagnostic Validity of Rorschach Test.—Fifty patients seen by one of the authors in consultation were submitted to the Rorschach test. The other writer made his diagnosis entirely on these tests without any clinical knowledge of the cases. Comparison between the Rorschach and the clinical evaluations of the cases showed a very high degree of correspondence, and the authors conclude that the Rorschach test possesses a definite diagnostic value.

Aspects of the Brain Potentials.—Studies of the electric potentials in the cerebral cortex of 80 insane patients showed great variation in the frequency, pattern, and amplitude of the e.e.g. led off from the occipital area. In a group of manic depressives the predominant rhythm was between 20–40 per second. Following amytal narcosis there was a return to the slow rhythm in two of these patients immediately after recovery from narcosis for a short interval during which time the patients were free from symptoms of hypomania. With clinical improvement there was a permanent return to the normal alpha rhythm. In a group of schizophrenic patients the potentials developed under amytal anæsthesia were not markedly different from those seen before the amytal administration. They are said to have been characterized by very fast rhythms and bursts of activity with frequencies as high as 500 per second. In a subsequent discussion Jasper suggested that the high frequencies which he had also observed in schizophrenics were due to muscular and not to cortical activity. The paper is not

illustrated and the details given are insufficient for the reader to form any opinion as to the nature of the very fast rhythms which the authors claim to have also observed in conditions of hyperpnœa.

Ketogenic Diet in Epilepsy in Children.—From 1922–1936 the Ketogenic diet was used at the Mayo Clinic to treat 501 children with epilepsy: 31 per cent. of the cases with idiopathic epilepsy were rendered free from attacks and 16 per cent. became definitely improved when Ketogenic diet was given an adequate trial. Approximately 11 per cent. of cases of symptomatic epilepsy were free from fits when treated with ketogenic diet. Eight per cent. of the patients with idiopathic epilepsy whose fits had been abolished by Ketogenic diet had a recurrence of the attacks within 7 years following the last attack. They had resumed normal diet for periods between 2–4 years when the attack occurred. After more than 7 years no recurrences were reported.

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The Path of Seen Movement as a Function of the Vector-Field. J. F. Brown and A. C. Voth. 543.
Conditioned Discrimination: the Development of Discrimination with and without Verbal Report. E. R. Hilgard, A. A. Campbell, and W. N. Sears. 564.
A Study upon the Recall of Pleasant and Unpleasant Words. P. T. Young. 581.
Whole and Part Methods in Puzzle Solution. L. W. Crafts and H. M. Kohler. 597.
Changes in Skin Resistance and Affective Tone. N. W. Shock and C. H. Coombs. 611.
The Affective Value of Pitch and Tempo in Music. K. Hevner. 621.
The Insensitivity of the Cornea to Heat and Pain derived from High Temperatures. J. P. Nafe and K. S. Wagoner. 631.
The Dependency of Cold upon Vascular Action Studies with Nerve Block. J. P. Nafe and K. S. Wagoner. 636.
The Effect of Adaptation upon Vascular Reactions to Thermal Stimuli. J. P. Nafe and K. S. Wagoner. 645.
The Reliability of Introspection in Emotion. W. A. Hunt. 650.
Minor Studies from the Psychological Laboratory of Clark University. Visual Acuity as a Function of Intensity and Exposure-Time. C. H. Graham and C. Cook. 654.
The Latency of Visual After-Effects as a Function of the Intensity of Illumination on an Adjacent Retinal Region. J. Levine and C. H. Graham. 661.

Vol. 51. No. 1. January 1938

The Problems of Mental Disability in England. M. Bentley. 1.
The Neurological Level of the Factors Underlying Time-Errors. G. Kreezer. 18.
*Studies in Vibratory Sensibility. L. H. Cohen and S. B. Lindley. 44.
Time as a Factor in the Recognition of Visually Perceived Figures. M. A. Seidenfeld. 64.
The Effect of Pattern upon Simultaneous Letter-Span. H. Woodrow. 83.
The Principle of Closure in Terms of Recall and Recognition. J. J. Tiernan. 97.
The Rôle of Incentive in Conditioning and Extinction. W. K. Brogden, E. A. Lipman, and E. Culler. 109.
*The Effect of Pain upon Peripheral Blood Volume. J. P. Nafe and K. S. Wagoner. 118.
The Discrimination of Color Mass. B. R. Philip. 127.
*The Relation between Psychosis and Physical Type: A Statistical Study. E. F. Wells. 136.
The Optimal Muscular Tensions for Various Performances. G. L. Freeman. 146.
Studies from the Psychological Laboratory of Vassar College.
The Comparative Efficiency of Intensity, Perspective, and the Stereoscopic Factor in Producing the Perception of Depth. M. F. Washburn and C. Wright. 151.
An Apparatus for Measuring Changes in Bodily Resistance. A. R. Lauer and D. E. Anderson. 156.
A Nerve-Action Potential Amplifier. F. Henry. 159.
Apparatus for the Study and Treatment of Enuresis. O. H. Mowrer. 163.

Vibratory Sensibility.—A somewhat complicated and cumbersome apparatus has been used to test the sensation of vibration. It has been shown that the diameter of the surface of stimulator in contact with the skin is of importance in obtaining a low threshold for this form of sensation. Studies were also made after the anæsthetization of the skin. (E. A. C.)

Pain and Peripheral Blood Volume.—It has been shown that the sensation of pain gives rise to vasoconstriction. There is a relationship between the degree of vasoconstriction and the intensity of stimulus. (E. A. C.)

Psychosis and Physical Type.—Physical measurements have been made in 50 cases of dementia præcox and 50 cases of manic-depressive psychosis. No reliable differences were found between the measurements in these two groups. The author concludes that his studies do not substantiate Kretschmer's theory of distinct physical types or Kretschmer's correlation between bodily type and psychosis. (E. A. C.)

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Cornell Studies in Dynasomatic Psychology. M. Bentley. 203.
A Descriptive Study of Somatic Objects. H. C. Werner. 225.
An Experimental Approach to the Self-Object. M. S. Ryan. 235.
An Experimental Study of Anticipation. 253.
Progressive Change in Simple Action: a Study of Learning. S. G. Longwell. 261.
Mathematical Objects and Symbolizing. T. A. Ryan. 283.
A Psychological Consideration of Analogy. M. L. Lemmon. 304.
Retrospect and Prospect. M. Bentley. 357.

Ontogeny of Embryonic Behavior in Aves. XII. Stages in the Development of Physiological Activities in the Chick Embyro. Z. Y. Kuo. 361.
The Influence of Social Facilitation on Motor Performance at Different Levels of Intelligence. 379.
*The Effect of Pain upon Systemic Arterial Blood Pressure. J. P. Nafe and K. S. Wagoner. 390.
Sex Differences in the Perception of Color Mass. B. R. Philip. 398.

Pain and Systemic Blood Pressure.—The authors have been able to demonstrate that stimulation of the skin with a pin or heat is invariably followed by a rise in systemic pressure, though neither cardiac nor respiratory rate is affected. (E. A. C.)

ANNALES MÉDICO-PSYCHOLOGIQUES

Vol. 95. Part 2. No. 5. December 1937.

*Les Formes Cliniques du Caféisme Cérébral. (Clinical forms of caffein intoxication.) P. de Fortunié and Belfils. 725.
Hallucination et Champ Visuel. (Hallucination and the visual field.) F. Morel. 742.
Sur les Gommes du Cerveau au Cours de la Paralysie Générale Traitée par la Malaria. (On gumma of the brain in cases of general paralysis treated by malaria.) J. Dretler. 758.

Proceedings of the Société Médico-Psychologique.
A Propos de l'Écriture en Miroir. (On mirror writing.) P. Abely and M. Rancoule. 769.
Épilepsie Psychique Post-Traumatique Associée à un Syndrome d'Amnésie Continue. (Post-traumatic psychical epilepsy associated with a syndrome of continuous amnesia.) M. L. Marchand, Mme. Gaultier, and M. R. Stora. 776.
Deux Paralysies Générales Infantiles. (Two cases of infantile general paralysis.) M. Brissot and J. Maillefer. 780.
Syndrome de Korsakoff au Cours d'une Crise de Rhumatisme Aigu. (Korsakoff syndrome occurring during an attack of acute articular rheumatism.) Capgras, J. Lhermitte, Ajuriaguerra, and Dousinet. 794.
Sur la Méthode de D. Anglade pour l'Étude de la Neurologie. (Anglade's method for the study of Neuroglia.) R. Anglade, M. Rougean, and P. Royer. 800.
Deux Jumeaux Déments Précoces. (Twins suffering from dementia præcox.) S. Rousset and M. G. Daulmazon. 806.
Troubles de la Mémoire Post-Traumatique de Type Particulier. Confabulation et Amnésie Systématique. (Post-traumatic disturbances of memory. Confabulation and systematic amnesia.) S. Rousset and M. G. Daulmazon. 810.
Pleurs de Sang au Début de l'Accès Épileptique. (Tears of blood at the onset of an epileptic fit.) J. Picard. 824.

Clinical Forms of Caffein Intoxication.—The principal signs of caffein intoxication are : (*a*) Tremor found in 60 per cent. of the cases, which may persist for some weeks after coffee-drinking has ceased. Tremor is very rapid, chiefly affects the hands and rarely the tongue and face. (*b*) Hyperæsthesia, paræsthesia, pruritus. (*c*) Vertigo and gastric disturbances. Often intense anorexia. (*d*) Anaphrodisia ; abortion has also been observed. (*e*) Disturbances of the vegetative nervous system, e.g. tachycardia, vasomotor disturbances, dyspnœa, polyuria. (*f*) Cerebral symptoms : lessening of the sensation of fatigue, intellectual facilitation, insomnia, symptoms of drunkenness resembling alcoholism. The authors consider that the abuse of coffee-drinking only causes pathological symptoms in subjects with a constitutional susceptibility to caffein poisoning. Excessive coffee-drinking can be suppressed without deprivation symptoms supervening. Suppression leads to the disappearance of all the symptoms due to coffee poisoning.

Vol. 96. Part 1. No. 1. January 1938.

*Désordre de la Fonction Hypnique et Hallucinations. (Disorders of sleep and hallucinations.) J. Lhermitte. 1.
*La Technique Actuelle du Test Psycho-Diagnostique de Rorschach. (The technique in current use for the Rorschach test.) M. Monnier. 15.
Traitement de la Schizophrenie par le Pentamethylentetrazol (cardiazol). (Cardiazol treatment of schizophrenia.) H. Delgado. 23.

Proceedings of the Société Médico-Psychologique.
Remarques Cliniques sur la Periode Précomateuse au Cours du Traitement des États Schizophreniques par l'Insulinothérapie. (Clinical observations on the precomatose period during insulin therapy.) Y. Porch'er and M. Leconte. 66.
Forme Cerebrale Évolutive Anormale d'une Syphilis Héréditaire. (Abnormal cerebral development in the case of a hereditary syphilitic.) Marchand, Stora, and Mme. Gaultier. 70.
Kinesie de Jeu chez les Idiots. (Movements of idiots during play.) Mme. Bonnafous-Serieux, Guiraud, H. Ey.
Syndrome de Korsakoff Alcoolique Aigu. Mme. Bonnafous-Serieux. 74.
Hallucinations combinées de la Vue et de l'ouie chez un Enfant de 3½ Ans. (Combined hallucinations of vision and hearing in a child 3½ years old.) Mme. S. Leconte-Lorsignol. 74.

Disorders of Sleep and Hallucinations.—Two cases of vascular mesencephalic lesions with visual hallucinations are described. In both cases the hallucinations corresponded with persistent hypersomnia. The author finds similar visual hallucinatory incidents in cases of cataplexy. He is led by these considerations to integrate in a common psychophysiological mechanism both sensory hallucinations, inhibition of tonus, and paroxysmal attacks of sleep. He considers that these symptoms are all attributable to a disturbance of the integrated functions that are concerned with sleep.

Technique for the Rorschach Test.—A valuable survey of the technique of the Rorschach method serves as an introduction to the revised edition of the "Psychodiagnostik." The author concludes with two important counsels. First, he considers that it is absolutely indispensable for the evaluation of this test for the observer to know precisely the intellectual level of the subject. He recommends an additional examination of each patient by the Binet-

Simon or other method of intelligence testing. Secondly, he warns against the terms "introversion" or "extroversion" as employed by Rorschach; he considers that these schematizations do not necessarily correspond either to the results of the test or to the clinical data. The test should be brought back to its initial objective character and only purely descriptive terminology employed.

Vol. 1. No. 2. February 1938.

*Les "Kinesies de Jeu" chez les Idiots. (The choreo-athetoid movements associated with play.) Mme. Bonnafous-Serieux and H. Ey. 145.
Sur les Modifications Post-Traumatiques du Systeme Nerveux chez les Enfants et les Adolescents. (Post-traumatic changes in the nervous system in children and adolescents.) N. Oseretzky and Z. Stcheglova. 158.
*Le Traitement des Complications Nerveuses de l'Alcoolisme par la Strychnine. (The treatment of alcoholic disturbances of the nervous system by strychnine.) P. Cossa, H. Bougeant, M. Puech, and P. Sassi. 167.

Proceedings of the Société Médico-Psychologique.
Accès Maniaque au Cours d'une Maladie de Basedow. (Attack of mania in a case of exophthalmic goitre.) Laignel-Lavastine, Escalier, H.-M. Gallot, and H. Mignot. 188.
Psychose Hallucinatoire Chronique et Syphilis. (Chronic hallucinatory psychosis and syphilis.) R. Anglade, P. Royer, and M. Rougean. 192.
Sideromanie et Dipsomanie chez un Desequilibre. (Dipsomania and railway-phobia in a psychasthenic.) R. Stora and Mme. Gaultier. 197.
Diplegie Cerebrale chez un Enfant Atteint d'Idiote Congeniatale et d'Epilepsie. Foyers Pseudo-Kystiques Multiples Cerbraux et Protuberantiel. (Cerebral diplegia in a child suffering from congenital idiocy and epilepsy. Multiple pseudocystic lesions of the cerebrum and bulbar regions.) L. Marchand, M. Brissot and J. Maillefer. 200.
Claustromanie, Coprochesie, et Trichophorie par Aboulie Psychasthenique. (Claustromania, coprophobia, and hirsutism in a case of psychasthenic abulia.) P. Courbon and Mme. Buvat-Pochon. 207.
Manies de Veuvage. (Manias of Widows.) Mme. Bonnafous-Serieux and H. Ey. 225.
Essai d'Interpretation des Reactions Maniaques Survenues après une Emotion Triste (Manie après Veuvage). (Attempt to interpret maniacal reactions supervening on a sad emotion. Mania after widowhood.) X. Abely and M. Leconte. 232.
Tumeur sous-frontale bilaterale à Symptomatologie Affective. État Psychologique normal après Ablation de la Tumeur et des Deux Poles Frontaux. (Bi-lateral sub-frontal tumour causing affective symptomatology. Normal psychological state after ablation of the tumour and the two frontal poles.) H. Baruk, M. David, and H. Askenasy. 241.
Injections Intrarachidiennes de Vitamine B_1 dans la Psycho-Polynevrite de Korsakow. (Intrathecal injections of vitamin B_1 in Korsakoff's psychosis.) E. Martimor and P. Neveu. 242.

Choreo - Athetoid Movements. — The authors consider that in cases of mental deficiency exhibiting choreo-athetoid movements it is possible to distinguish a system of movements connected with playful or narcissistic tendencies. The movements are occasional; they always arouse interest and are accompanied by euphoria. They appear suddenly during periods of inactivity. They are inhibited by voluntary activity.

Treatment of Alcoholic Disturbances by Strychnine.—The intensive treatment of alcoholism with strychnine, first published by Lutin in 1873, has been revised after fifty years and its value endorsed by numerous authors. The writers consider that there is no form of alcoholic disturbance of the nervous system that cannot be successfully treated by strychnine. They call attention to the great tolerance of alcoholics for strychnine and give as much as 0·02 gm., but never more than 0·03 gm., in the 24 hours. Not only are both mental and neurological symptoms favourably affected, but during treatment by strychnine all desire for alcohol disappears. The authors proceed to discuss the possible mechanism of strychnine action in relation to alcoholism. They consider that as in barbiturate poisoning the administration of strychnine tends to correct a heterochronism of the peripheral and central chronaxies produced in this case by alcohol.

Vol. 1. No. 3. March 1938.

*Les Troubles de la Sensibilité à la Douleur dans la Paralysie Générale. (Disturbances of pain sensation in general paralysis.) R. Nyssen and R. Busschaert. 289.
Les Délires Secondaires au Traitement de la Paralysie Générale. (Secondary delirium occurring in the treatment of general paralysis.) A. Requet and J. Turin. 298.
*Recherches sur les Hormones Hypophysaires dans Certaines Psychoses. (Studies on pituitary hormone in certain psychoses.) M. Cahane and T. Cahane. 311.
Soixante Ans de Psychose Maniaco-Dépressive. (60 years of manic-depressive psychosis.) M. Olivier and Mme. Chevalier-Gazaniol. 321.

Proceedings of the Société Médico-Psychologique.
Sur l'Évolution des Délires d'Influence. Trois degrés de Déficit Résiduel. (On the evolution of delusions of compulsion.) J. Vie and M. Villemez. 327.
Hyperkinésie gesticulaire de Démence. (Gesticulatory hyperkinesia and dementia.) P. Courbon and Mme. Buvat-Pochon. 333.
Tremblement de Type Pithiatique, céphalées avec Bouffées Confusionelles, Perturbations Sympathiques Cervicales (d'Origine Traumatique). Guérison par le Venin de Cobra. (Hysterical tremor, headaches with confusional crises, disturbances of the cervical sympathetic of traumatic origin. Cured with cobra venom. H. Baruk and R. Mathon. 339.
Troubles Abasiques Hystériformes chez un Parkinsonien Post-Encéphalitique. (Hysterical abasia in a case of post-encephalitic Parkinsonism.) P. Sivadon and P. Queron. 345.
Automutilation à But Thérapeutique chez un Dément Présénile à Forme Dépressive. (Auto-mutilation with a therapeutic design in a case of depressed presenile dementia.) P. Sivadon and P. Queron. 347.
Résultats du Traitement de 111 Cas de Paralysie Générale par le Stovarsol Sodique. (Results of treatment of 111 cases of general paralysis with sodium stovarsol.) L. Marchand. 349.
La Thérapeutique Convulsivante de la Démence Précoce par le Pentaméthylènetétrasol (Cardiazol). (Convulsant therapy of dementia præcox with

cardiazol.) R. Anglade, P. Royer, and M. Rougean. 357.

Les Manifestations Motrices chez l'Enfant, de la Naissance à Trois Mois. (Movements exhibited by infants from birth to three months.) M. Bergeron. 367.

Psychose Hallucinatoire Curable de la Ménopause et Pousée Hypertensive. (The curable psychoses of the menopause and crises of hypertension.) P. Chatagnon, P. Scherrer, and C. Chatagnon. 373.

Pain Sensation in General Paralysis.—The sensory system of 102 cases of general paralysis was examined. Seventeen of these cases were untreated, the remaining 85 cases had been treated with malaria not less than some months before the examination. Of the 17 cases examined before malarial treatment, eight showed a diminution of sensibility to pain of varying degree. Of the 85 patients who had undergone malarial treatment, 65 showed a varying amount of hypoalgesia. Three patients appeared to have complete analgesia ; this did not, however, persist through repeated examinations in two of the cases ; the third case was permanently analgesic. Loss of the deep reflexes was present in 21 of the 102 cases, and of this group 86 per cent. were definitely hypoalgesic. Of the 81 cases with normal deep reflexes analgesia was present in 71 per cent. There was no definite correlation between the degree of mental impairment and the occurrence of analgesia.

Pituitary Hormone in Certain Psychoses.—An examination of 30 cases of affective psychosis showed either thyroid hypertrophy or hyperthyroidism in 40 per cent. The authors were induced by these observations to look for the presence of the thyrotropic pituitary hormone in the urine. In nine cases showing signs of thyroid hypertrophy the thyrotropic hormone was found in the urine of six cases. Twelve cases of mania with thyroid hypertrophy were examined for the melanophore-expanding hormone. Four cases gave positive results. The reaction was negative in the urine of two cases of confusional insanity and four cases of schizophrenia. A positive melanophore reaction was observed in two castrates.

ARCHIVES DE NEUROLOGIE DE BUCAREST

Vol. 2. No. 1. 1938.

Les Connaissances Actuelles sur l'Hystérie. (Present-day conception of hysteria.) D. Paulian. 3.

L'Hystérie et le Diencéphale. (Hysteria and the diencephalon.) A. Radovici. 9.

Considerations sur le Mécanisme et le Traitement du Mutisme Hystérique. (The mechanism and treatment of hysterical mutism.) A. Dimolesco. 15.

Quelques Remarques sur les Rapports de l'Hystérie et de la Schizophrénie. (Some observations on hysteria and schizophrenia.) H. Claude. 23.

Note sur le Mécanisme Psycho-Physiologique de Certains Das de Pythiatisme. (On the psycho-physiological mechanism in cases of Pythiatism.) Laignel-Lavastine. 30.

Le Problème de l'Hystérie et ses Dilemmes. (The problem of hysteria.) J. Froment. 33.

L'Hystérie et les Fonctions Diencéphaliques. (Étude Neurologique.) (Hysteria and the functions of the diencephalon.) C. Vincent. 46.

L'Hystérie et le Pythiatisme Vus par un Psychiâtre. (Hysteria and pythiatism as seen by a psychiatrist.) E. Gelma. 52.

Quelques Remarques sur la Position due Problème de l'Hystérie. (Some remarks on the problem of hysteria.) H. Baruk. 69.

Le Problème de l'Hystérie. (The problem of hysteria.) A. Salmon. 77.

Vol. 2. No. 2. 1938.

Physiothérapie Électrique des Paraplégies. (Electro-physio-therapy of paraplegia.) D. Paulian and I. Bistriceanu. 102.

Les Variations de la Tension Artérielle dans l'Hémiplégie sous Influence de l'Effort Thérapeutique. (Blood pressure in hemiplegia and the effect of effort,) E. Marinesco-Bojoïo. 107.

Contributions à l'Étude du Corps Jaune et de ses Indications. (Contribution to the study of the corpus luteum.) D. Paulian and E. Copcea. 113.

L'Érotisme Délicteux de l'Homme d'Âge Mûr et Non Dément. (Criminal sexuality in the adult and mentally healthy man.) E. Gelma. 117.

Über die Bedeutung des diastolischen Blutdruckes für die Beurteilung Hyperthyreosen. (Diastolic blood pressure in the diagnosis of hyperthyroidism.) K. Hitzenberger. 123.

Le Rôle des Noyaux Hypothalamiques dans la Vie Psycho-Affective. (The rôle of the hypothalamic nuclei in psycho-affector symptoms.) A. Salmon. 132.

L'Hystérie au Point de Vue Biologique. (Biological viewpoint of hysteria.) V. M. Buscaino. 147.

ARCHIVES OF NEUROLOGY AND PSYCHIATRY

Vol. 38. No. 4. October 1937.

*Meningiomas : Origin, Divergence in Structure and Relationship to Continuous Tissues in Light of Phylogenesis and Ontogenesis of the Meninges, with Suggestion of a Simplified Classification of Meningeal Neoplasms. J. H. Globus. 667.

*Pathologic Features of Multiple Sclerosis and Allied Conditions. G. B. Hassin. 713.

*A Proposed Mechanism of Emotion. J. W. Papez. 725.

Mode of Onset of Epilepsy. J. L. Fetterman and V. R. Hall. 744.

*Sympathectomy in Man : Its Effect on the Electrical Resistance of the Skin. C. P. Richter and M. Levine. 756.

*Association of Carotid Sinus Reflexes with Syncope and Convulsions. A. S. Freedburg and Le R. H. Sloan. 761.

*Necrotizing Encephalitis simulating Tumor of the Brain Clinically and due to Necrotizing Angiitis. N. A. Levy. 775.

*Water Metabolism in Relation to Convulsions. T. T. Stone and H. Chor. 798.

Meningiomas.—In a long and lavishly illustrated paper the author discusses the origin and diversity of structure of the meningioma in the light of the phylogenesis and ontogenesis of the meninges. A new simplified classification of the meningiomas is offered. This is based on the identification of each variety with some dominant cell form and cell organization which characterizes a stage in the development of the meninges. (R. M. S.)

Multiple Sclerosis and Allied Conditions.—According to Hassin the fundamental feature of multiple sclerosis is that the changes are not confined to the plaques but are diffuse and scattered throughout the entire central nervous system; that is, even areas apparently normal exhibit histological changes. The nerve fibres are attacked primarily and directly, without the intermediation of the blood vessels. (R. M. S.)

Mechanism of Emotion.—It is suggested that the hypothalamus, the anterior thalamic nuclei, the gyrus cinguli, the hippocampus, and their interconnections constitute a harmonious mechanism which may elaborate the functions of central emotion, as well as participate in emotional expression. (R. M. S.)

Sympathectomy in Man.—Cervical sympathectomy (removal of the stellate and first thoracic ganglia) in 10 patients consistently produced a large increase in the electrical resistance of the skin of the palms of the hands. Cervical sympathectomy produced a smaller and less consistent increase in the electrical resistance of the skin on the backs of the hands. Lumbar sympathectomy (removal of the second, third, and fourth lumbar ganglia) in two patients produced similar changes in the resistance of the skin on the soles and the backs of the feet. It was concluded that readings of the electrical resistance of the skin should be as useful for the study of sympathetic disturbances in man as they have proved to be in monkeys and cats. This method has the advantage over others in that it requires little time and can be repeated at frequent intervals, with little inconvenience either to the patient or to the observer. (R. M. S.)

Carotid Sinus Reflexes.—The authors consider that in a number of patients with attacks of weakness, vertigo, atypical epilepsy, and vague epileptiform seizures, the carotid sinus reflex may be ætiologically responsible. The diagnosis is based on the repeated induction of these attacks by pressure on the carotid sinus and the inability to produce them after procainization. If the carotid sinus is found to be hyper-irritable in patients with the symptoms described, local pathological conditions in the region of the carotid sinus should be removed and denervation of the carotid sinus should be considered. (R. M. S.)

Necrotizing Encephalitis.—A case is presented of massive necrotizing encephalitis simulating a tumour of the brain, which was due to widespread necrotizing angiitis of unknown ætiology. Cases of unusual inflammatory vascular disease of the brain reported in the literature, particularly periarteritis nodosa and the "arteritis" of unknown ætiology described by Harbitz, and their relationship to the present case are discussed. (R. M. S.)

Water Metabolism in Relation to Convulsions.—From a study of 18 young adult epileptics who were subjected to hydration and dehydration, the conclusion is reached that it remains to be proved whether a restriction of fluid intake can diminish or prevent the occurrence of convulsions. (R. M. S.)

Vol. 38. No. 5. November 1937.

*Sensory Functions of the Optic Thalamus of the Monkey (*Macacus rhesus*): Symptomatology and Functional Localization investigated with the Method of Local Strychninization. J. G. D. de Barenne and O. Sager. 913.
*Electrical Stimulation of the Cortex Cerebri of Cats: Responses Elicitable in Chronic Experiments through Implanted Electrodes. S. L. Clark and J. W. Ward. 927.
*Phenylpyruvic Oligophrenia: Introductory Study of Fifty Cases of Mental Deficiency associated with Excretion of Phenylpyruvic Acid. G. A. Jervis. 944.
"Cold Pressor Test" in Tension and Anxiety: a Cardiochronographic Study. B. V. White and E. F. Gildea. 964.
Curve of the Sugar Content of the Blood following Encephalography: Comparison with the Usual Curve for Dextrose Tolerance. M. Scott. 985.
*Studies in Diseases of Muscle. I. Metabolism of Creatine and Creatinine in Progressive Muscular Dystrophy. A. T. Milhorat and H. G. Wolff. 992.
*Encephalomyelitis complicating Measles. N. Malamud. 1,025.

Sensory Functions of Optic Thalamus of the Monkey.—This paper is concerned with an investigation of the sensory functions of the thalamus of the monkey by means of the method of local strychninization. (R. M. S.)

Electrical Stimulation of Cortex Cerebri of Cats.—By a method of permanently implanting electrodes on specific points of the cortex cerebri which allows recovery from anæsthesia and the effects of operation, cortical points in cats were stimulated at

intervals over long periods with and without anæsthesia. The experiments demonstrate that the same response can be elicited from the same cortical point from day to day and argue against any marked instability of cortical points except under the temporary influence of transient physiological states. The influence of an anæsthetic on the response to repeated stimulation of a cortical point appears to be expressed by the depression or elimination of the effects (of facilitation) on neighbouring cortical areas before the depression or abolition of the response from the point under the electrode. The picture is similar to that obtained in the responses to successively weaker stimuli applied to the same point. No evidence of pain as a primary response to cortical stimulation was observed, though some evidence of sensory effects was found. The method allows the production of epileptiform seizures at will in the "normal" animal. (R. M. S.)

Phenylpyruvic Oligophrenia.—A preliminary study of 50 cases of mental deficiency associated with excretion in the urine of phenylpyruvic acid enabled the author to exclude any relation of endogenous agencies to the ætiology of the disease, whereas the family incidence of the disease suggests that a genetic mechanism is of ætiological significance. Statistical elaboration and critical analysis of the genetic figures afford justification for regarding the condition as determined by a single recessive gene substitution. From a biochemical point of view the condition appears to be an error of the metabolism of phenylalanine, consisting essentially of a failure to oxidize further a normal catabolite, phenylpyruvic acid, which consequently is excreted as such in the urine. On what appear to be wholely inadequate grounds the author considers that extrapyramidal symptoms are a characteristic component of the disease. (R. M. S.)

Diseases of Muscle.—The principal conclusion from this study is that the creatine output and creatine tolerance would appear to be an expression of the total mass of improperly functioning muscle and not, *per se*, an index of the amount of wasted muscle or the primary site of the disease. (R. M. S.)

Encephalomyelitis complicating Measles.—Two cases of encephalomyelitis complicating measles are reported, illustrating, respectively, the acute phase and the end-stage of the disorder. The condition is interpreted as a true encephalitis. The fundamental differences between measles encephalitis and multiple sclerosis are discussed. (R. M. S.)

Vol. 38. No. 6. December 1937.

*Epidemic Encephalitis : a Follow-Up Study of 266 Cases. W. L. Holt. 1,135.
*Degeneration of the "Boutons Terminaux" in the Spinal Cord. An Experimental Study. W. C. Gibson. 1,145.
*Factors producing Lumbar Cerebrospinal Fluid Pressure in Man in the Erect Posture. T. von Storch, E. A. Carmichael, and T. E. Banks. 1,158.
Hypertensive Apoplexy and Its Causation. W. H. Chase. 1,176.
*Facial Tic in relation to Injury of the Facial Nerve. An Experimental Study. H. A. Howe, S. S. Tower, and A. B. Duel. 1,190.
*Roentgenologic Study of Orientation of the Pineal Body. I. Comparison of the Proportional and the Graphic Method in Absence of Tumor of the Brain. W. W. Fray. 1,199.
A Clinicopathologic Study of Astrocytomas. R. W. Waggoner and K. Löwenburg. 1,208.
*Changes in the Brain in Plexectomized Dogs, with Comments on the Cerebrospinal Fluid. G. B. Hassin, E. Oldberg, and M. Tinsley. 1,224.
Entostoses within the Calvarium : Survey of Skulls in the Warren Museum of the Harvard University Medical School. M. M. Canavan. 1,240.
*Arterial Supply of Lateral Parolivary Area of the Medulla Oblongata in Man. L. Alexander and T. H. Suh. 1,243.
*Oxygen Metabolism inSchizophrenia. R. G. Hoskins. 1,262.

Epidemic Encephalitis.—Of 78 patients first seen in the acute stage at the Boston Psychopathic Hospital whose condition was diagnosed as epidemic encephalitis, 11·5 per cent. are alive and without known sequelæ after from 10 to 16 years. The prognosis for lasting recovering appears not to be altered appreciably by the prominence of mental symptoms during the acute attack, for other authors have recorded similar rates of recovery for patients in general hospitals.

Of 90 patients with sequelæ observed again after from 10 to 17 years, 7·7 per cent. are apparently recovered. For 72 patients with sequelæ who came under observation more recently, but after a longer elapsed time since the acute attack, the rate of recovery is 1·4 per cent. Children with behaviour disorders constitute the only group with sequelæ of epidemic encephalitis in whom improvement may reasonably be expected. (R. M. S.)

Degeneration of Boutons Terminaux in Spinal Cord.—*Boutons terminaux* are generally believed to be the end processes of axons synapsing on the body and dendrites of a nerve cell. In this paper the author presents pictorially observations based on a study at regular intervals of some stages of degenerating *boutons terminaux* in the cat after section of the spinal cord. (R. M. S.)

Lumbar S.C.F. Pressure in Man in Erect Posture.—From observations made in patients in the National Hospital it is concluded that lumbar cerebrospinal fluid pressure in man, measured in the erect

sitting position, is determined by the intradural venous pressure existing at the lumbar level. This venous pressure is conditioned by the hydrostatic effect of the column of blood below the right auricle, the pressure existing within the right auricle, and the rate of flow of the blood. Since the difference in pressure responsible for the rate of flow is very small, the lumbar venous pressure is approximately equal to the sum of the vertical distance between the point of puncture and the pressure in the right auricle. (R. M. S.)

Facial Tic in Injury of Facial Nerve.—Anatomical and physiological studies were made on the facial muscles of 27 rhesus monkeys in which facial paralysis and subsequent recovery had taken place after interruption of the facial nerve. After an average of 44 days function began to return in the paralysed muscles, and after from 52 to 79 days purposeless tic-like movements made their appearance. These tics involved various portions of the facial musculature and were synchronous with purposeful movements elsewhere in the facial field. From the demonstration, first, of a phenomenon similar to the axon reflex in the regenerated facial field and, second, of actual branching of axons at the site of injury, it was concluded that single regenerated axons had by branching come to supply widely separated muscles and that an irrevocable functional union had taken place between muscles which do not normally contract simultaneously. The tics which follow injuries to the facial nerve in man are discussed in the light of these observations. (R. M. S.)

Roentgenologic Study of Orientation of Pineal Body.—The position of the pineal body as revealed by roentgenology is of importance in the diagnosis of brain tumour and cerebral atrophy, and in this paper an account is given of a simple method of localization, which is based on strict proportionality of the measurements of the anteroposterior and the vertical diameter of the skull. (R. M. S.)

Changes in Brain in Plexectomized Dogs.—Dandy's conclusion that the origin of the cerebrospinal fluid is largely, if not exclusively, in the choroid plexus is challenged by Hassin and his co-workers, who have repeated his experimental work on animals. Using Dandy's technique with slight modifications, the choroid plexus was removed from 30 dogs which were allowed to live from 2 days to 8 months. The experimental evidence indicated that cerebrospinal fluid forms in the ventricles but not from the choroid plexus. Its source is the cerebral tissue fluids drained by the ventricles through the connecting Virchow-Robin channels. (R. M. S.)

Arterial Supply of Lateral Parolivary Area of Medulla Oblongata in Man.—The lateral parolivary area of the human medulla oblongata is supplied in its anterior third (half or fourth) by the anterior artery of the lateral parolivary fossa (a branch of the basilar artery). In its posterior two-thirds (half or three-fourths) it is supplied by the posterior artery of the lateral parolivary fossa (a branch from the posterior inferior cerebellar artery), supplemented by one or two intermediate arteries of the lateral parolivary fossa (branches from the vertebral artery).

In case of absence of either the anterior or the posterior artery of the lateral parolivary fossa associated with rudimentary development of the intermediate artery or arteries, either the anterior or the posterior artery of the lateral parolivary fossa supplies the entire lateral parolivary area. (R. M. S.)

Oxygen Metabolism in Schizophrenia.—Studies bearing on the problem of oxygen metabolism in schizophrenia which have been carried out in the Worcester State Hospital during the last decade indicate that defective oxygen metabolism is an important feature in this psychosis. (R. M. S.)

ARCHIV FÜR PSYCHIATRIE UND NERVENKRANKHEITEN

Vol. 107. No. 2. November 1937.

*Über die Indikation zur Insulinschockbehandlung der Schizophrenie. (The indication for treatment by insulin in schizophrenia.) R. Lemke. 223

Die Frage der Schwangerschaftsunterbrechung bei Geisteskranken und psychisch Abnormen. (The interruption of pregnancy in mental disorders.) T. Stuppler. 240.

Familiäre amyotrophische Lateralsklerose. (Familial amyotrophic lateral sclerosis.) G. Rütenik. 248.

*Beiträge zur Histopathologie der Paralysis agitans. (Histopathology of paralysis agitans.) A. Juba and A. Szatmari. 265.

Über optische Sinnestäuschungen als epileptisches Äquivalent bei traumatischer Schädigung des Hinterhauptlappens. (Visual hallucinations as an epileptic equivalent in injury of the occipital lobe.) A. Szatmari. 290.

*Über vasomotorische Reaktionen und Wärmeregulation im katatonen Stupor. (Vasomotor reactions and heat regulation in catatonic stupor.) R. Jung and E. A. Carmichael. 300.

Über die Pathogenese der Lyssa. (Pathogenesis of rabies.) I. Schükrü-Aksel. 339.

Jakob Haringer. Eine psychopathologische Untersuchung über die Lyrik. (Mit Hinweisen auf Hermann Hesse und Max Herrmann.) (Jakob Haringer. A psychopathological study on the lyric with reference to Hermann Hesse and Max Herrmann.) R. Flinker. 347.

*62. Wanderversammlung der südwestdeutschen Neurologen und Psychiater am 22. und 23. Mai 1937 in Baden-Baden. (62nd meeting of South-West German neurologists and psychiatrists on May 22—23, 1937, at Baden-Baden.) 400.

*Über eine Frühform der Alzheimerschen Krankheit (28. bis 39. Lebensjahr) mit Olivensklerose und hochgradiger Atrophie der Stammganglien (terminaler Chorea). (An early form of Alzheimer's disease (28-39 years of age) with sclerosis of the olive and intensive atrophy of the basal ganglia (terminal chorea).) H. Kufs. 431.

Insulin Treatment of Schizophrenia.—Report on 46 cases treated by the insulin method at the Jena clinic. The results fall considerably short of those reported from Austria and Switzerland, but agree with more recent experiences of Mauz and Lehmann-Facius. Social remission was reached only in 26 per cent. of the total cases, of which 50 per cent. were not acute. There were four fatalities. After-histories made personally by the author on untreated schizophrenics several years after the beginning of the illness showed that 34 per cent. had resumed work. The author believes that insulin influences only those cases which would improve without treatment, though the treatment appears to shorten the schizophrenic attack considerably. He advocates the use of the encephalogram in order to select cases for treatment. Considerable enlargement of the sulci and the ventricles in the encephalogram precludes favourable results from treatment. (A. M.)

Histopathology of Paralysis Agitans.—Histological investigation of five cases. The globus pallidus is the predominant site of the lesions, while striate body and substantia nigra are only slightly involved. Senile plaques and argentophil fibril changes are scarce and inconsistent in cortex and basal ganglia. It is therefore doubtful whether the disease is to be regarded as a local manifestation of senility. Discussion of the various localizatory conceptions of tremor and acinetic syndrom. (A. M.)

Vasomotor Reactions in Catatonic Stupor.—In eight cases of catatonic stupor it is shown by means of finger plethysmography, thermoelectric registration of skin and rectal temperature and observation of the heat reaction that the inclination to peripheral vasoconstriction, coolness of the extremities, cyanosis, and œdema may be explained by the immobility of the patient. There is no positive indication of any real abnormality of the peripheral and central vegetative nervous system. This negative result, though it clarifies previous controversial statements, should not discredit the belief in the organic nature of psychosis. (A. M.)

The Meeting of the South-West German Neurologists and Psychiatrists.—Many papers deal with the influence of age on the appearance of mental and nervous disease. The paper of Schaltenbrand on the age incidence of brain tumour, of Zillig on general paralysis from the same viewpoint may be specially mentioned. Marchionini dealt with the disappearance of diastase in syphilitic spinal fluids, and he observed the same phenomenon in extracts of cortex taken from G.P.I. brains. (A. M.)

Alzheimer Disease.—Histological description of a case whose chief features are already indicated in the above title. Discussion of similar cases in the literature. Particular importance is attached to v. Braunmuehl's theory of ageing. (A. M.)

Vol. 107. Nos. 3 and 4. December 1937.

*Die Zentrale Haubenbahn und ihre Bedeutung für das extrapyramidal-motorische System. (Central tegmental tract and its significance in the extrapyramidal system.) E. Weisschedel. 443.

Zur Frage des "persönlichen Tempos" bei den Depressiven. (Personal tempo in depression.) F. Ferreira. 580.

Beiträge zum Problem der Entfremdungserlebnisse bei Hirnkranken. (Problem of depersonalization in brain disease.) E. Pappenheim. 599.

Zur Symptomatologie der Delirien bei occipitoparietalen Herden. (Symptomatology of delirium in occipito-parietal lesion.) A. Auersperg und A. Flach. 616.

Zur Anatomie und Patho-Physiologie des vorstehend beschriebenen Falles. (Anatomy and Pathophysiology of the preceding case.) O. Pötzl. 636.

Zur Genese der haptischen Halluzinationen im Verlaufe von intoxikativen Psychosen. (Origin of haptic hallucinations in the course of toxic psychoses. P. Büchler and J. Szepesi. 651.

Internistische Erfahrungen bei Durchführung der Malaria- und Pyriferbehandlung. (Medical observations in the course of malaria and pyrifer treatment.) H. Vock. 658.

*Über die Cholinesterase im Blut bei Myasthenie und Morbus Parkinson. (Cholinesterase in the blood in myasthenia and Parkinson's disease.) E. Pichler. 669.

Central Tegmental Tract.—Careful historical review on all we know about this tract. Personal investigation consists of a complete myelin series and confirms the previous observations that the main part of the tract originates in the red nucleus, with a small remnant coming from the globus pallidus. The previous name, thalamo-olivary tract, is therefore wrong. Besides the fibres going to the olive there are definite connections with the substantia reticularis of the mid-brain, pons, and medulla, from which centrifugal fibres originate to end probably in the spinal anterior horns. From the olive efferent extrapyramidal impulses are carried via Hellweg's bundle or the olivary-cerebellar connections. The central tegmental tract is in man the most important descending extra-pyramidal fibre system, while the rubro-spinal tract is of minor significance. In contrast to the pyramidal, the extrapyramidal pathway consists of short interrupted systems. This may signify the possibility of substantial modification of the impulses while travelling to the peri-

phery. The clinical significance of the central tegmental tract is but little understood at present. (A. M.)

Cholinesterase in Myasthenia and Parkinson Disease.—Even the smallest amounts of prostigmin inhibit the esterase. In myasthenia the content of esterase in the blood is diminished. In Parkinson's disease the content is normal, but increased after muscular activity in contrast to the findings in normal persons and those afflicted with myasthenia. (A. M.)

Vol. 107. No. 5. March 1938.

Über Kleinhirnveränderungen bei multipler Sklerose. (Cerebellar changes in disseminated sclerosis.) W. Ammerbacher. 675.

*Histologischer Gehirnbefund nach Insulinkoma. (Histological findings in the brain after insulin coma.) F. Kobler. 688.

Pupillenstörungen bei Hirntumoren und ihre Beziehungen zu anderen Schädigungen des Sehorgans. (Pupil changes in brain tumours and their relation to other changes of the visual apparatus.) P. Demmler. 701.

Lidnystagmus nach doppelseitigen Kaltspühlungen der Ohren. (Nystagmus of the eyelid after bilateral aural stimulation by cold.) H. Brunner. 711.

Zur Klinik der sog. Arachnitis adhaesiva. (Clinical symptomatology of arachnitis adhæsiva.) G. E. Störring. 717.

*Beiträge zur Kenntnis der Kerne des Hypothalamus bei Säugetieren. (Study of the hypothalamic nuclei in mammals.) H. Koikegami. 742.

*Bemerkungen über die Chloruro-Kristallisation des Liquors. (Remarks on the "Chloruro-Crystallization" in the cerebrospinal fluid.) H. Selzer. 775.

Zur Psychopathologie des Zwischenhirns (Thalamus und Hypothalamus). (Psychopathology of the diencephalon (thalamus and hypothalamus.) G. E. Störring. 786.

Histology of Insulin Death.—After a short review of previous cases the author describes a personal observation. Besides slight glial and mesodermal reaction, the main feature was a vacuolic degeneration of the ganglion cell nuclei, particularly marked in the cornu ammonis and medulla. This change is interpreted as being caused by direct toxic effect of insulin overdosage upon the cell nucleus. The possibility of a vascular pathogenesis, however, is not entirely ruled out. (A. M.)

Mammalian Hypothalamus.—Careful and well-illustrated comparative anatomical study carried out on representative species of lower and higher mammals. The most important result is the repudiation of Gruenthal's statement that there is in the ascending phylogenetic scale a decrease of the number of nuclei. According to the author, Gruenthal was misled by his arbitrary separation of the nuclei. In higher mammals the supraoptic nucleus consists of three instead of two parts as in lower species; the paraventricular nucleus of two instead of one. Nuclei tubero-mammillaris and laterales tuberis are found only from the apes onwards, etc. The author tries to bring some order into the conflicting terminology without avoiding the creation of new names. (A. M.)

"Chloruro-Crystallization" in C.S.F.—This method advocated by Bergauer *et alia* as a test for pathological spinal fluid has no diagnostic significance according to the authors. (A. M.)

BRAIN

Vol. 60. No. 4. December 1937.

*Epilepsy: A Paroxysmal Cerebral Dysrhythmia. F. A. Gibbs, E. L. Gibbs, and W. G. Lennox. 377.

*Somatic Motor and Sensory Representation in the Cerebral Cortex of Man as studied by Electrical Stimulation. W. Penfield and E. Boldry. 389.

*Compression of the Chiasma. Optic Nerves and Optic Tracts by Intracranial Aneurysms. G. Jefferson. 444.

*Observations on Dystrophic Myotonica. O. Maas. 498.

The Exogenous Cellular Content of the Neurohypophysis in Man under Pathological Conditions. H. J. Wade. 525.

*Hydrocephalic and Focal Cerebral Symptoms in Relation to Thrombophlebitis of the Dural Sinuses and Cerebral Veins. C. P. Symonds. 531.

Epilepsy.—Epilepsy is due to defective control of cerebral rhythm. During a seizure the potentials recorded by the electro-encephalograph are of high voltage and of different frequency from the normal 8 to 20 a second. In "grand mal" a frequency of 25 to 30 occurs, in psychomotor attacks 3 or 4 a second, while in "petit mal" fast and slow waves alternate. The last variety are also present during subclinical attacks, while the two former may precede the seizures. The abnormal rhythms usually originate in the frontal area, but not infrequently in the parietal or occipital areas. Epileptics tend to show a greater variability of rhythm than do normals and show an exaggeration of the normal changes in rate. Phenobarbital and bromides slow the rhythm and are indicated for "grand mal," but not for psychic variants. Abnormal rhythms are increased by insulin and decreased by glucose and CO_2. Mental activity tends to inhibit "petit mal" rhythms. (J. D.)

Somatic and Sensory Representation.—The human cortex was stimulated over 800 times at 163 operations on conscious individuals and the sensory and motor phenomena elicited were recorded. Despite an individual variation the pre- and post-central gyri showed the generally accepted

topography. In disagreement with Foerster, the motor area of the tongue was placed below the jaw. Stimulation just above the lip area caused vocalization. The topography of the post-central gyrus accurately followed that of the pre-central and there was an overlap between the two, sensation being elicited from the pre-central gyrus and movement from the post-central. Tingling and numbness were the commonest sensations. Pain was only felt 11 times. There was no localization of the quality of the sensation. Excluding epileptiform movements, which continue after stimulation has ceased, no motor activity occurred from stimulation of cortex other than that of the post- and pre-central gyri, and a small area just anterior to this for adversive eye movements. Any mass or turning movements due to stimulation of 6a beta were epileptic in nature. Ipsilateral movement or sensation did not occur, but bilateral movements and sensations occurred in the face and tongue. There was no evidence of a vasomotor centre on the convexity of the hemispheres, and visceral or emotional responses were rare. Secondary facilitation displaced the sensory responses as well as the motor. (J. D.)

Compression of the Chiasma.—From a group of 53 cases of intracranial aneurysm the author has selected 12 basal aneurysms which had affected the visual pathways. They have been roughly divided into four groups. In three cases the aneurysm affected the optic radiation or the striate cortex by ischæmia following thrombosis. Precise localization of the lesion was not possible. In one group the optic tract was compressed by an aneurysm of the posterior cerebral artery. The optic nerves were compressed in two cases, one of which was verified as being due to an aneurysm of the subclinoid internal carotid which filled the middle fossa. The chiasma was compressed three times by anterior cerebral aneurysms and three times by those of the internal carotid, the former acting from above and the latter from below. In addition to these chiasmal cases 71 were collected from the literature and the field defects fully discussed. The commonest were monocular central scotomata or blindness, alone or in association with temporal hemianopia in the contralateral eye, homonymous, hemianopia, and bitemporal hemianopia. Sudden fluctuations in vision were common and the discs were usually atrophic. Severe pain appearing abruptly and roughly contemporaneous with disturbance of vision is characteristic of aneurysms, but they may closely mimic a benign tumour. X-ray demonstration of calcification in the walls of the aneurysm, destruction of the sella, or widening of the sphenoidal fissure are useful aids in diagnosis, while angiographic demonstration is conclusive. In most instances carotid ligation is to be recommended. (J. D.)

Dystrophia Myotonica.—158 members of 57 families in which dystrophia myotonica occurred were examined. Only four were found to be without suspicion of the disease. The material is analysed to show that it is transmitted by dominant inheritance and that fraternal anticipation occurs. (J. D.)

Hydrocephalic and Focal Cerebral Symptoms in Thrombophlebitis.—Five further cases of otitic hydrocephalus are reported. Evidence is presented showing that the hydrocephalus is due to defect in the C.S.F. absorption mechanism and this has been attributed to thrombosis of the superior longitudinal sinus. This conclusion is based on the demonstration that thrombosis can spread from the lateral sinus to the superior longitudinal sinus and that thrombosis of the superior longitudinal sinus, not otitic in origin, can produce a similar picture. Support for this view is also found in the fact that of the reported cases 65 per cent. were secondary to the right ear, and this corresponded fairly well with percentage in which the superior longitudinal sinus joins the right lateral sinus rather than the left. Several cases illustrative of the association of cerebral venous and superior sinus thrombosis are also reported. (J. D.)

Vol. 61. No. 1. March 1938.

*Increased Spontaneous Activity produced in Monkeys by Brain Lesions. C. P. Richter. 1.
*Spontaneous Hæmatomyelia : a Short Review and a Report of Cases illustrating Intramedullary Angioma and Syphilis of the Spinal Cord as Possible Causes. J. C. Richardson. 17.
*On Normal Discs in Patients with Chiasmal Lesions. A. A. McConnell and A. J. Mooney. 37.
*The Neurone as Studied by Micro-Incineration. L. Alexander. 52.
*The Origin of the Fall in the Pressure of the Cerebrospinal Fluid after its Artificial Elevation by Jugular Occlusion. T. H. B. Bedford. 62.
*Vascular Lesions in the Brain-Stem and Occipital Lobe occurring in Association with Brain Tumours. M. T. Moore and K. Stern. 70.
*Observations on the Pathways transmitting the Sensation of Taste. H. G. Schwartz and G. Weddell. 99.
A Case of Primary Generalized Amyloid Disease with Involvement of the Nerves. S. de Navasquez and H. A. Treble. 116.

Increased Spontaneous Activity in Monkey with Brain Lesions.—Nineteen monkeys were kept in special cages so designed that a record of the amount of movement could be obtained. A total of 46 cerebral operations were performed. It was concluded that activity is definitely controlled through the prefrontal cortex, particularly area 9, and also through the striatum. Lesions of these structures produced an increase in activity. (J. D.)

Spontaneous Hæmatomyelia.—Four cases

of hæmatomyelia are reported and two were proven at autopsy to be complications, one of syphilitic meningomyelitis and the other of a developmental angioma. The term " spontaneous " is used to indicate that the hæmorrhage was the first indication of the disease. Trauma is of little ætiological importance in this condition. (J. D.)

Normal Discs in Chiasmal Lesions.—A report of six cases of verified tumours involving the chiasma unassociated with optic atrophy. In most instances there were marked field defects ; five of the six tumours lay behind the chiasma, suggesting that the absence of atrophy may be of diagnostic import. It appears that pallor of the discs is due to involvement of the optic nerves rather than the chiasma or tracts. (J. D.)

The Neurone studied by Micro-Incineration.—Sections of various parts of the nervous system were incinerated at 650° C. Ganglion cells contain rich deposits of heat-resistant mineral ashes in their nucleolus and Nissl bodies. The nucleus, axone hillock, and axis cylinder are almost free of ash. The mineral ash content of the cerebellar and cerebral cortex is greater than that of the isocortex and basal ganglia and there are characteristic appearances corresponding to various fields of the cerebral cortex. The mineral content of the cells is greater during development and apparently has some relationship to growth and metabolic activity. (J. D.)

Fall in Pressure of C.S.F. after Jugular Occlusion.—It had previously been found that the C.S.F. pressure returned to its original level within an hour. This phenomenon was investigated, using a method which permitted the C.S.F. pressure to be maintained at any desired level and which also permitted the measurement of the flow of fluid into or out of the subarachnoid space. When the pressure was kept at its high level after jugular occlusion a continuous inflow was observed. When the pressure was kept at the original level a rapid displacement of fluid occurred and subsequently either a very slow outflow or no flow was observed. It was concluded that the fall in C.S.F. pressure after jugular occlusion was due to increased absorption in those venous sinuses least influenced by the occlusion. (J. D.)

Vascular Lesions in Brain Stem and Occipital Lobe.—A clinical and pathological description of 14 cases of supratentorial tumour associated with distant vascular lesions. In five there was thrombosis of one or both post-cerebral arteries attributable to compression by the herniated uncus. In two of these and in the remaining nine hæmorrhages were found in the mid-brain and pons. These are fatal and result from an increase in systemic blood-pressure produced reflexly by sudden changes in intracranial tension. A raised supratentorial pressure and herniation at the tentorium which cause congestion in the mid-brain act as predisposing causes. (J. D.)

Pathways transmitting Taste.—It was found in four cases that section of the trigeminal root did not affect taste. In three cases section of the sensory root of the fifth nerve was followed by loss of taste on the homolateral two-thirds of the tongue. Two of these had evidence of injury to the facial nerve, but one was uncomplicated except for section of the greater superficial petrosal nerve. Three cases after excision of the chordo tympani had homolateral loss of taste, while in two cases it was preserved. It therefore appeared that the pathway for taste from the anterior two-thirds of the tongue was subject to variation. In some instances it was conveyed by the chorda tympani, otic ganglion, internal sphenoidal nerve, and greater superficial petrosal nerve to the geniculate ganglion, while in others by the chorda tympani and facial nerve. (J. D.)

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Vol. 6. No. 3. December 1937.

*Migraine in Children and the Mechanism of the Attack. H. A. Riley. 387.
*The Sense of Smell. XVI. A Comparison of the Results of Encephalography and Ventriculography and of Quantitative Olfactory Tests for the Localization of Supratentorial Tumors of the Brain. C. A. Elsberg. 403.
*The Value of Quantitative Visual Tests for the Localization of Supratentorial Tumors of the Brain. A Preliminary Report. C. A. Elsberg and H. Spotnitz. 411.
*Are Vision and the Olfactory Sense governed by the Same Laws ? A Comparison of the Results of Quantitative Functional Tests of Vision and of the Sense of Smell and Its Significance. C. A. Elsberg and H. Spotnitz. 421.
*A Comparison of the Symptoms and Signs of Intracerebral and Extracerebral Tumors involving the Temporal Lobes. R. T. Collins. 430.
Ganglion Cell Tumors of the Central Nervous System. A. Wolf and B. F. Morton. 453.
Encephalographic Appearance of an Intraventricular Epidermoid. C. G. Dyke and L. M. Davidoff. 489.
*Thresholds of Vibratory Sensibility as determined by the Pallesthesiometer. A Study of Sixty Normal Subjects. R. W. Laidlaw and M. A. Hamilton. 494.
Acute Demyelinating Encephalomyelitis following Respiratory Disease. C. Davison and S. Brock. 504.
*Pathological Study of a Group of Cases sometimes referred to as Polyneuritis. W. M. Honeyman. 519.

Posterior Inferior Cerebellar Artery Thrombosis J. D. Spillane. 529.
*The Reactions of Patients to Encephalography. An Analysis of One Thousand Cases. S. S. Bohn. 540.
*A Note on the Appearance of Meningioma in Tissue Culture. A. Wolf and W. M. Honeyman. 569.
*Pyogenic Infections within the Vertebral Canal. M. M. Campbell. 574.

Migraine in Children.—Eight patients under the age of 13 with migraine were observed and the salient features of the disease at this age are described. Current theories of the mechanism of the attack are discussed. (J. D.)

Sense of Smell.—Sixty-one supratentorial brain tumours were investigated by ventriculography and the author's olfactory tests. In 77 per cent. olfactory tests were sufficiently positive to indicate the side of the lesion and in 80 per cent. of these the tests gave adequate information as to the region to be exposed at operation. (J. D.)

Visual Adaptation Tests in Brain Tumours.—A preliminary report on the value of dark adaptation tests in the localization of brain tumours. In 19 verified cases the visual tests gave adequate information as to the side of the lesion and as to whether or not it involved the frontal lobe in 80 to 90 per cent. The test is based on a previous demonstration that dark adaptation is mainly a central process. It is defective in the eye opposite to the side of the lesion. Lesions of the frontal lobe cause an inversion of the adaptation times to continuous and flickering light. (J. D.)

Comparison of Vision and Olfaction.—The formulæ expressing olfactory fatigue and visual adaptation are compared and shown to be the same. As similar laws govern both it is probable that they are dependent on a cerebral mechanism. (J. D.)

Intra- and Extra-Cerebral Temporal Tumours.—The symptoms and signs of 108 neoplasms affecting the temporal lobes have been investigated; 74 were intracerebral, while 34 were extra-cerebral. Only 50 per cent. of cases had symptoms definitely referable to dysfunction of the temporal lobe. Contralateral motor and sensory disturbances and visual field defects were the most important localizing signs. A comparison of the extra- and intracerebral neoplasms showed that the former have a longer course and a greater incidence of auditory disturbances and mental aberrations. Trigeminal neuralgia occurred only in this group. The intracerebral neoplasms caused more field defects and more marked contralateral motor and sensory signs. Drowsiness and stupor were confined to this group. (J. D.)

Vibration Sense.—The amplitude threshold of vibration sense was investigated on different regions of the body and the results on a group of young normal subjects compared to those on older subjects. The finger-tips were uniformly the most sensitive areas, while areas of obesity raised the threshold. Marked variations in threshold were found on adjacent spots and the threshold was increased with age, especially in the lower part of the body. (J. D.)

Pathological Study of Cases called Polyneuritis.—Four cases of severe polyneuritis showing mainly motor disturbances were investigated clinically and at post-mortem. No significant pathological abnormality was discovered. (J. D.)

Reactions to Encephalography.—The reactions following encephalography in 1,000 consecutive cases are reported. Atropine is recommended as a preoperative drug, while morphine is contra-indicated. The symptoms and their duration are enumerated. Two fatalities occurred and both patients had brain tumours. Failure of the ventricles to fill occurred in 10 per cent., but the percentage was much higher in the group with brain tumours and in those cases where a general anæsthetic was used. (J. D.)

Meningioma in Tissue Culture.—Fragments of four meningiomas were cultured. Three of these showed typical mesodermal elements, while the fourth showed a rapid growth of solid columns of cells which were probably of arachnoidal origin. (J. D.)

Pyogenic Infections of Vertebral Canal.—A brief review of the literature, together with a report of eight cases of purulent infection of the extra-dural space. The cases are characterized by pyrexia, local tenderness, root pains, and signs referable to dysfunction of the spinal cord, especially sphincter disturbances. The mode of spread may be either hæmatogenous or direct through the intervertebral canals. Laminectomy should be performed as an emergency measure to allow drainage. (J. D.)

L'ENCEPHALE

Vol. 32. No. 4. November 1937.

Un Cas de Dystrophie Intermédaire entre le Syndrome d'Apert et celui de Hurier.
A Case of Dystrophy Intermediate between the Syndrome of Apert and that of Hurier. G. Muyle and L. van Bogaert. 169.
De Quelques Psychoses Schizophreniformes dans l'Enfance.
Schizophrenic Psychosis in Children. Dr. Langfeld. 183.

JOURNAL OF ANATOMY

Vol. 72. No. 2. January 1938.

*Meninges and Cerebro-Spinal Fluid. L. H. Weed. 181.
*The Exogenous Cellular Content of the Neurohypophysis in Man under Normal Conditions. H. J. Wade. 216.
A Technique for Operations in the Hypothalamo-Hypophysial Region of the Rabbit. G. W. Harris and G. T. Popa. 226.
Further Remarks on the Terminations of Nerves in Human Teeth. O. W. Tiegs. 234.
The Development of the Pars Membranacea Septi in the Human Heart. P. M. B. Rodgers. 247.
The Early Stages of Replacement of the Semilunar Cartilages of the Knee-joint in Rabbits after Operative Excision. R. Walmsley and J. Bruce. 260.
The Effects of Prolonged Vestrogeneric Stimulation, on the Prostrate of the Rhesus Monkey. S. Zuckerman. 264.
The Effects of Male Hormone on a Mature Castrated Rhesus Monkey. S. Zuckerman and A. S. Parkes. 277.
An Outline of Elliot Smith's Contributions to Neurology. H. H. Woollard. 280.
Two Skulls with Absence of the Premaxilla. D. E. Derry. 295.
Anomalous Heart. G. I. Boyd. 299.
The External Characters of a Second Australian Fœtus. F. Wood-Jones. 301.

Meninges and C.S.F.—This article is too long to abstract, but should be read by all interested in the C.S.F. (E. A. C.)

Exogenous Cellular Content of Neurohypophysis.—A study has been made of 75 pituitary glands. It has been found that there are more basophil cells in the male than in the female gland. The invasion by basophil cells may reach considerable proportions in perfectly healthy subjects. (E. A. C.)

Vol. 72. No. 3. April 1938.

The Primitive Form of Epiphysis in the Long Bones of Tetrapods. R. W. Haines. 323.
A Study of Spinal Cord Development in Silver-Stained Sheep. L. G. Scharpenberg and W. F. Windle. 344.
Observations on the Cervix Uteri and the Urethra of Monkeys. O. C. Sandys and C. Zuckerman. 352.
Some Observations on the Fœtal Vessels of the Human Placenta, with an Account of the Corrosion Technique. P. Bacsich and C. F. V. Smout. 358.
The Development of the Supra-Umbilical Portion of the Anterior Abdominal Wall. G. M. Wyburn. 365.
The Investigation of Cell Size with Special Reference to the Human Lymphocyte. D. Mainland and B. K. Coady. 374.
Lymphatics of the Skin, with a Note on Lympathic Watershed Areas. G. Forbes. 399.
The Cervical Vertebræ of the Australian Native. F. Wood-Jones. 411.
The Course of the Incisor Branch of the Inferior Dental Nerve in Rodents and Some Observations on the Nerve Supply of the Pulp. E. Ridehalgh and D. Stewart. 416.
Abnormal Development of the Brain in the 8-mm. Pig Embryo. J. S. Baxter and J. D. Boyd. 422.
The Morphology and Permeability of the Roof of the Fourth Ventricle in Some Mammalian Embryos. H. Cohen and S. Davies. 430.
A Rare Malformation of the Hand. R. S. Stacey. 456.
The Prepùtial Glands of British Muridæ. L. H. Jackson. 458.

JOURNAL BELGE DE NEUROLOGIE ET DE PSYCHIATRIE

Vol. 37. No. 10. October 1937.

Paralysie Bulbaire chez une Tabétique. (Bulbar paralysis in a tabetic.) P. Divry and P. Houyez. 619.
Signification Diagnostique de la Ventriculographie pour la Tumeur du Corps Calleux. (Diagnostic significance of ventriculography in tumour of the corpus callosum.) T. de Lehoczky. 625.
Contribution au Syndrome Polyradiculonévritique de Guillain-Barré. (Contribution to the polyneuritic syndrome of Guillain-Barré.) L. Laruelle and L. Massion-Verniory. 635.
Le Traitement Convulsivant de la Démence Précoce. (The convulsive treatment of dementia præcox.) A. Leroy. 646.

Vol. 37. No. 11. November 1937.

Introduction au Problème de l'Encéphalite Vaccinale. (Introduction to the problem of post-vaccinal encephalitis.) L. van Bogaert. 675.
L'Encéphalite Post-Vaccinale aux Pays-Bas. (Post-vaccinal encephalitis in the Pays-Bas.) F. S. van Bouwdijk-Bastiaanse. 685.
Encéphalite Post-Vaccinale Mortelle. Démonstration Anatomique. (Fatal post-vaccinal encephalitis. Pathological demonstration.) E. Kokken. 696.
Un Cas de Myélite Transverse Post-Vaccinale. Guérison. Présentation du Malade. (A case of post-vaccinal transverse myelitis with recovery.) J. Mage. 703.
Considérations Cliniques sur l'Encéphalite Post-Vaccinale Infantile. Deux Cas Personnels. (Discussion of clinical features of post-vaccinal encephalitis in children.) M. Meunier. 706.

Vol. 37. No. 12. December 1937.

*Tumeur Cérébrale Parvicellulaire se Propageant dans l'Espace de Virchow-Robin. (Small-celled cerebral tumour propagating in the Virchow-Robin spaces.) J. Mage and H. J. Scherer. 731.
La Paralysie Agitante à Caractère Héréditaire et Familial. (Heredo-familial paralysis agitans.) R. Dellaert, R. Hyssen, and L. van Bogaert. 747.
Réactions Vestibulaires Anormales Observées au Cours de Lésions Intramédullaires Cervicales et Cervico-Dorsales en Evolution. (Abnormal vestibular reactions observed in the course of cervical and cervico-dorsal intramedullary lesions.) J. Helsmoortel and L. van Bogaert. 752.
Dystrophies Cutanées Multiples à Caractère Hérédi-

taire, Polynévrite et Troubles Mentaux. (Maltiple cutaneous lesions of hereditary character with polyneuritis and mental disorder.) H. Baonville, J. Ley, and J. Titeca. 760.

Neurinome de l'Acoustique avec Symptomatologie Vestibulaire Anormale. (Acoustic neuroma with abnormal symptomatology.) F. Crabbé. 768.

Small-celled Tumour in Perivascular Spaces.—Mage and Scherer describe a tumour affecting the basal ganglia on one side in a man of 50 years, with wide dispersion in the perivascular spaces, though the meninges are only slightly invaded. It is concluded that the tumour is primary, and of a characteristic type of which three earlier cases have appeared in the literature, all with identical situation, histology, and clinical features (profound mental changes, change in character, finally a rapidly progressive hemiplegia with sensory change). The histogenesis of the small cells is undetermined. (D. D.-B.)

Vol. 38. No. 1. January 1938.

*La "Glioblastome en Plaques." (On multiple patches of glioblastoma.) H. J. Scherer. 1.

L'Influence des Excitations Algiques sur la Tension Artérielle chez les Paralytiques Généraux. (The effect of painful stimulation on the blood pressure of general paralytics.) R. Nyssen, R. Busschaert, and L. Asaert. 18.

Sur les Indications de la Thérapeutique Convulsivante. (On the indications for convulsant therapy.) P. Verstraeten. 28.

Un Cas de Saturnisme Chronique Tardif, Suite de Blessures de Guerre (Fragments de Grenade) ayant simulé, successivement, une Sclérose en Plaques, puis une Polynévrite avec Ulcère Peptique. (A case of chronic lead poisoning following war wounds from grenade fragments, having simulated successively disseminated sclerosis, then polyneuritis with gastric ulcer.) E. de Savitsch. 32.

Le Traitement de la Sitiophobie par le Na-Cl Hypertonique et le Scopochloralose. (The treatment of negativism by hypertonic saline and chloralose-scopolamine.) T. Rubbens and F. Wallez. 39.

Gliablastoma.—Scherer describes the pathology of a case of multiple sharply delineated patches of gliablastoma in the brain stem and cerebral white matter. The patches resemble an inflammatory demyelinating lesion and in places frank gliablastoma with a tendency to grow in concentric bands. A neoplastic change supervening on a disseminate inflammatory lesion is the explanation that is favoured. (D. D.-B.)

Vol. 38. No. 2. February 1938.

*The Anatomical Basis of the Thalamic Syndromes. A. E. Walker. 69.

Un Syndrome d'Ataxie Cérébelleuse Progressive avec Oligophrénie chez Deux Jeunes Israélites Polonais. (A syndrome of progressive cerebellar ataxia with oligophrenia in two young Polish Jews.) M. Maere and G. Muyle. 96.

*Le Problème de la Localisation et des Ataxies Corticales. (The problem of the localization of cortical ataxias.) M. A. Gerebtzoff. 108.

Syndrome du Trou Occipital par Abcès du Lobe Frontal. (Syndrome of the foramen magnum produced by frontal abscess.) L. Laruelle and L. Massion-Vernjory. 133.

Thalamic Syndromes.—Walker has written an excellent summary in English of his work on the constitution of the thalamic nuclei and their relationship with spinal, cerebellar, and cortical projection systems. The disorders resulting from damage to different parts of the thalamus are discussed in relation to this recent work. (D. D.-B.)

Cortical Ataxias.—An anatomical discussion of the cerebello-preparietal projection. (D. D.-B.)

Vol. 38. No. 3. March 1938.

*Essai sur un Groupe Épidémique de Cas de Polyradiculonévrite avec Dissociation Albumino-Cytologique du Liquide Céphalo-Rachidien (Type de Guillain et Barré), chez l'Enfant et chez l'Adulte. (Discussion of an epidemic group of cases of infective polyneuritis with albumino-cytological dissociation in the cerebrospinal fluid (Guillain-Barré syndrome) in the child and in the adult.) L. van Bogaert, F. Philips, J. and M. A. Radermaker, and T. Verschraegen. 151.

Un Cas de Polyradiculonévrite avec Dissociation Albumino-Cytologique et Oedème de la Papille. (A case of infective polyneuritis with albumino-cytological dissociation and œdema of the papilla.) P. van Gehuchten. 212.

Diplègie Faciale Curable. (Curable facial diplegia.) P. Martin and J. Titeca. 217.

A propos d'un Cas Anormal de Poliomyélite Antérieure Aiguë chez une Enfant. (An abnormal case of acute anterior poliomyelitis in a child.) Prof. Dereux. 228.

Quelques Remarques sur l'Étiologie de la Maladie de Guilain-Barré. (Some remarks on the ætiology of the Guillain-Barré syndrome.) A. Biemond. 231.

Syndrome de Guillain et Barré et Varicelle chez un Enfant. (Guillain-Barré syndrome and chicken-pox in a child.) Clément and Kételbant. 240.

Polyneuritis.—Van Bogaert and his collaborators conclude that the affection described in 1916 by Guillain and Barré can be considered as a morbid entity, probably of infectious origin. An acute poly-radiculitis with rise of protein in the C.S.F. without increase in cells has a very favourable prognosis, though some cases occasionally came to autopsy. The dissociation between increase of albumen and of cells in the C.S.F. is seen in less degrees in other more chronic types of neuritis, and even alcoholic neuritis in slight degree. Cases of polyneuritis without dissociation may also have a favourable course. Nevertheless, it is worth while maintaining the clinical distinction of the complete syndrome of Guillain and Barré. Dr. Biemond notes occasional mild pleocytosis in association with the condition. (D. D.-B.)

JOURNAL OF CLINICAL INVESTIGATION

Vol. 16. No. 6. November 1937.

The Bisulphite Binding Power of the Blood in Health and in Disease, with Special Reference to Vitamin B_1 Deficiency. F. H. L. Taylor, S. Weiss, and R. W. Wilkins. 833.
The Estimation of the Subcutaneous Tissue Pressure by a Direct Method. G. S. Burch and W. A. Sodeman. 845.
Clinical Studies of the Blood Volume. III. Changes in Blood Volume, Venous Pressure, and Blood Velocity Rate in Chronic Congestive Heart Failure. J. G. Gibson and W. A. Evans, Jr. 851.
The Measurement of Glomerular Filtration, Creatinine, Sucrose, and Urea Clearances in Subjects without Renal Disease. A. W. Winkler and J. Parra. 859.
The Measurement of Glomerular Filtration. The Creatinine, Sucrose, and Urea Clearances in Subjects with Renal Disease. A. W. Winkler and J. Parra. 869.
A Convenient Method for the Determination of the Approximate Cardiac Output in Man. J. S. Donal, Jr. 879.
Chronic Pyelonephritis and Arterial Hypertension. A. M. Butler. 889.
Allergy and Desensitization in Tuberculosis. H. S. Willis and C. E. Woodruff. 899.
Relation of Serum Calcium to Serum Albumen and Globulins. A. B. Gutman and E. B. Gutman. 903.
Studies in Serum Proteins. I. Identification of a Single Serum Globulin by Immunological Means. Its Distribution in the Sera of Normal Individuals and of Patients with Cirrhosis of the Liver and with Chronic Glomerulonephritis. F. E. Kendall. 921.
A Quantitative Study of the Oxidation of Glucose in Normal and Diabetic Men. J. M. Sheldon, M. W. Johnston, and L. H. Newburgh. 933.

Vol. 17. No. 1. January 1938.

Chloride Depletion in Conditions other than Addison's Disease. A. W. Winkler and O. F. Crankshaw. 1.
The Effects on the Cardiovascular System of Fluids administered intravenously in Man. I. Studies of the Amount and Duration of Changes in Blood Volume. D. R. Gilligan, M. D. Altschule, and M. C. Volk. 7.
Studies of Gonococcal Infection.
III. A Comparison of the Bactericidal Properties of the Synovial Fluid and Blood in Gonococcal Arthritis. W. W. Spink and K. S. Keefer. 17.
IV. The Effect of Mucin on the Bacteriolytic Power of Whole Blood and Immune Serum K. S. Keefer and W. W. Spink. 23.
The Renal Excretion of Endogenous Creatinine in Man. Comparison with Exogenous Creatinine and Insulin. B. F. Miller and A. W. Winkler. 31.
The Effect of Epinephrin in Circulatory Collapse induced by Sodium Nitrite. R. W. Wilkins, S. Weiss, and F. W. Haynes. 41.
The Neutralization of Encephalitis Virus (St. Louis, 1933) by Serum. R. S. Muckenfuss, J. E. Smadel, and E. Moore. 53.
Respiratory Properties of the Arterial Blood in Normal Man and in Patients with Disease of the Liver. Position of the Oxygen Dissociation Curve. A. Keys and A. M. Snell. 59.
Observations on the Renal Threshold for Ascorbic Acid in Man. J. M. Faulkner and F. H. L. Taylor. 69.
The Distribution of Body Water and Electrolytes in Adrenal Insufficiency. H. E. Harrison and D. C. Darrow. 77.
*The Effect of Hyperthermia on the Distribution of Water and Electrolytes in Brain, Muscle, and Liver. H. Yannet and D. C. Darrow. 87.
The Metabolism of Blood Pigments in Pernicious Anæmia. K. Dobriner and C. P. Rhoads. 95.
The Excretion of Caproporphyrin I. following Hæmorrhage in Dogs. K. Dobriner and C. P. Rhoads. 105.
The Influence of Epinephrine on the Digital Arterioles of Man. A Study of the Vasoconstrictor Effects. T. J. Fatherree and E. V. Allen. 109.

Effect of Hyperthermia on the Brain.—Hyperthermia was shown to redistribute the water in brain from the cells to the extracellular fluid, without any change in the total water content, together with concentrations of K, P, and N within the cells. These changes occurred whether convulsions did or did not take place. (J. N. C.)

Vol. 17. No. 2. March 1938.

Urea Excretion at Low Urine Volumes. The Calculation of "Minimal" Urea Clearances. L. C. Chesley. 119.
The Excretion of Porphyrin in Refractory and Aplastic Anemia. K. Dobriner, C. P. Rhoads, and L. E. Hummel. 125.
The Effect of Artificial Fever and Specific Antiserum on the Organisms Present in Cases of Type III Pneumococcus Meningitis. M. F. Shaffer, J. F. Enders, and J. Wilson. 133.
The Flow of Blood in the Coronary Artery in Pathological Hearts. W. B. Kountz and J. R. Smith. 147.
Clinical Studies of the Blood Volume. IV. Adaptation of the Method to the Photoelectric Microcolorimeter. J. G. Gibson and K. A. Evelyn. 153.
Alkaline Tides. G. D. Barnett and F. E. Blume. 159.
A Note on the State of Calcium in High Protein Serum. H. I. Chu and A. B. Hastings. 167.
*Ascorbic Acid in Cerebrospinal Fluid. M. Pijoan, L. Alexander, and A. Wilson. 169.
The Concentration of Glucosamine in Normal and Pathological Sera. R. West and D. H. Clarke, with the technical assistance of E. M. Kennedy. 173.
A Study of the Blood Iodine in Childhood. G. J. Fashena. 179.
The Effects of Pressor Drugs and of Saline Kidney Extracts in Blood Pressure and Skin Temperature. E. M. Landis, H. Montgomery, and D. Sparkman. 189.

Ascorbic Acid in Cerebrospinal Fluid.—A reasonably accurate method for the assay of ascorbic acid in cerebrospinal fluid is described; the quantities found were almost identical with those occurring in blood plasma. (J. N. C.)

JOURNAL OF COMPARATIVE NEUROLOGY

Vol. 67. No. 3. October 1937.

*Sensory Nerve Endings on the Smooth Muscle of the Urinary Bladder. F. Kleyntjens and O. R. Langworthy. 367.
Development of the Brain of Amblystoma in Early Functional Stages. C. J. Herrick. 381.
*Thymonucleic Acid in Purkinje Cells. Y. T. Loo. 423.
*The Relative Vascularity of Various Parts of the Central and Peripheral Nervous System of the Cat and its Relation to Function. H. S. Dunning and H. G. Wolff. 433.
*The Superior Colliculus in Vision. E. E. Ghiselli. 451.
*Cell Fiber Ratios in the Vagus Nerve. R. L. Jones. 469.
*The Ratio of Myelinated to Unmyelinated Fibers in Regenerated Sciatic Nerves of *Macacus Rhesus*. H. A. Davenport, H. Chor, and R. E. Dolkart 483.
*Development of the Spinal Reflex Mechanism in Human Embryos. W. F. Windle and J. E. Fitz-Gerald. 493.

Sensory Nerve-Endings in Smooth Muscle of Urinary Bladder.—Medullated fibres leaving the spinal cord with the posterior sacral roots were found to end in arborizations upon the smooth muscle of the cat's bladder. Non-medullated fibres appear to have a part in the formation of these endings. These are believed to form the anatomical basis for the stretch reflex. Many simple motor nerve-endings were seen. (J. D.)

Thymonucleic Acid in Purkinje Cells.—Using Feulgen's stain the distribution of thymonucleic acid was studied in the Purkinje cells of rats of different ages. With increasing age the amount diminished in the nucleus, appearing at the same time in the cytoplasm, and then further diminishing in both as senile dissolution set in. The early stages of this progressive change are attributed to overgrowth of the cytoplasm in highly differentiated cells. (J. D.)

Vascularity of Nervous System of Cats.—The vascularity of different parts of the nervous system were determined histologically. A comparison of (1) the trigeminal ganglion, which has no terminal arborization and many cells; (2) the white matter of the hemisphere, which consists of axons; and (3) the superior cervical sympathetic ganglion, which contains cells and many synapses, showed that the vascularity was proportional to the number of synapses. This was confirmed by observations on different layers of the parietal cortex. It was concluded that this arrangement was necessary to permit a sufficient rate of oxidation in the very fine strands of the neurophil which have a great neural surface area. (J. D.)

Superior Colliculus in Vision.—Lesions of the superior colliculus in rats produced no loss in retention of either discrimination or change in visual acuity or brightness threshold. It was concluded that the superior colliculus alone did not mediate detail and brightness discrimination habits, but it was not yet possible to say if it could do so in the absence of the geniculostriate complex. (J. D.)

Cell Fibre Ratios in Vagus Nerve.—The number of fibres in the vagus nerve of the cat was measured proximal and distal to the nodose ganglion and the number of cells in the ganglion counted. The number of fibres in the proximal section was similar to the number of cells, but the distal trunk contained 17 per cent. more fibres. No evidence of branching or of synapses was found in the ganglion, all the cells being unipolar. It appeared, therefore, that the great majority of the fibres in the cervical vagus were processes of nodose ganglion cells, and that some of these cells were motor in function. (J. D.)

Fibre Ratios in Regenerated Sciatic Nerves.—The sciatic nerves of eight monkeys were studied six months after unilateral section and resuture of the sciatic nerves. The ratio of myelinated to unmyelinated fibres was more variable in the regenerated nerves and about four-fifths of the total number of fibres was restored. The maturity of the myelin sheaths varied greatly from nerve to nerve and from place to place in the same nerve. (J. D.)

Spinal Reflex Mechanism in Human Embryos.—Twenty-seven human embryos 5 to 8 weeks old were studied. The primary motor, primary sensory, and secondary neurons were present in the youngest specimens and the basic elements for a spinal reflex were present at 6 weeks. However, muscle development, central integrating mechanisms, and peripheral motor and sensory endings do not reach a functional stage until the eighth week. (J. D.)

Vol. 68. No. 1. December 1937.

A Study of the Surface Structure of the Brain of the Whale. (*Balænoptera physalus* and *Physeter catodon.*) F. A. Ries and O. R. Langworthy. 1.
The Lateral-Line System of the Teleost *Fundulus heteroclitus*. M. Denny. 49.
*Pericellular End-Bulbs in the Central Nervous System of Vertebrates. G. S. Phalen and H. A. Davenport. 67.
Further Experimental Studies of the Development of Lateral-Line Sense Organs in Amphibians observed in Living Preparations. L. S. Stone. 83.
The Structure of the Vertebrate Synapse. A Study

of the Axon Endings on Mauthner's Cell and Neighbouring Centers in the Goldfish. D. Bodian. 117.
The Nervous System of *Dolichoglossus Kowalevskyi*. W. N. Hess. 161.

Pericellular End-Bulbs.—Marked variation in size and number of the end-bulbs was found in different mammals and in different parts of the central nervous system. This variation suggested that their degeneration was not a suitable index for locating synaptic connections unless the degenerated tract was massive. (J. D.)

Vol. 68. No. 2. February 1938.

The Relative Influence of the Locus and Mass of Destruction upon the Control of Handedness by the Cerebral Cortex. G. M. Peterson and L. C. Fracarol. 173.
Localization in the Oculomotor Nuclei of the Goldfish. Z. Hadidian and M. S. Dunn. 191.
Development of the Cerebrum of Amblystoma during Early Swimming Stages. C. J. Herrick. 203.
*The Spinal Accessory Nerve in Human Embryos. A. A. Pearson. 243.
*Fiber and Cellular Degeneration following Temporal Lobectomy in the Monkey. R. W. Rundles and J. W. Papez. 267.

Spinal Accessory Nerve.—Seven human embryos 7 to 12 weeks of age were examined. The spinal accessory portion of the XIth nerve was found to originate in a group of cells in the ventral horn of the cervical cord down to C_8. Some of the fibres emerge directly, while others have a short ascending intramedullary course. Ganglia were found along the nerve and afferent fibres entering the spinal cord turned dorsally into the posterior funiculus. Other afferent fibres entered with C_1. (J. D.)

Temporal Lobectomy in the Monkey.—The brains of two monkeys were examined 7 and 26 months after bilateral temporal lobectomy. Commissural connections between the two lobes run in the corpus callosum and anterior commissure. The medial geniculate nucleus sends fibres to the cortex lying in the lower wall of the sylvian fissures, the parageniculate fibres lying more caudal. No direct connection exists between the pulvinar and temporal cortex, but the ventral nucleus of the thalamus is connected by corticofugal and corticopetal fibres with the subcentral and lower postcentral gyri. There is no temporal cortical projection tract to the pons. The degeneration in the striate cortex following interruption of the optic radiation is described and also certain connections of the tail of the caudate nucleus and putamen. (J. D.)

JOURNAL OF MENTAL SCIENCE

Vol. 84. No. 348. January 1938.

The Eighteenth Maudsley Lecture: Science and Psychiatry. F. Golla. 4.
*Huntington's Chorea: A Study of Thirty-Four Families. L. Minski and E. Guttmann. 21.
On Some Problems of Histological Diagnosis and Interpretation of Circulatory Disturbances in the Brain. A. Meyer. 97.
Schizophrenia-like Psychoses in Young Children. R. A. Q. Lay. 105.
The Control of Dysentery by Prophylactic Inoculation. K. C. L. Paddle. 134.
Insulin Treatment of Schizophrenia.
I. Recent Advances in Insulin Treatment. H. P. Strecker. 146.
II. Hypoglycæmic Treatment of Schizophrenia: Some Impressions. A. A. W. Petrie. 156.
Insulin Therapy: A Review, with Special Reference to the Mechanism of Cure. R. Freudenberg. 165.
Somnifaine Narcosis: Results of 40 Treatments in 38 Cases. J. Kearney. 177.
Some Developments, Legal and Administrative, in Mental Treatment. R. H. Curtis. 183.
Responsibility in Mental Disorder, with Special Reference to Algolagnia. W. N. East. 203.

Huntington's Chorea.—Thirty-four families have been studied. The members were examined for minor anomalies on both the psychological and neurological sides. The results were analysed. It is intended to amplify this report at a later date. (E.A.C.)

JOURNAL OF NERVOUS AND MENTAL DISEASE

Vol. 86. No. 4. October 1937.

*Malignant Tumors of the Nasopharynx. W. Needles. 373.
Comparative Intelligence Ratings in the Four Types of Dementia Præcox. C. E. Trapp and E. B. James. 399.
Hypnosis: a Rational Form of Psychotherapy in the Treatment of the Psychoneuroses. J. L. McCartney. 405.
Spontaneous Intraspinal Subarachnoid Hæmorrhage. H. B. Slavin. 425.
*The Nature of Tolerance to Ethyl Alcohol. H. Newman and J. Card. 428.

Malignant Tumours of Nasopharynx.—Thirty-five cases of nasopharyngeal malignancy are reported, together with a review of the literature. Stress is laid on the absence of local signs and the early involvement of the cervical glands and Eustachian tube. The growth may spread through the base of the skull to produce a picture similar to that of a basal meningioma or

aneurysm. These tumours are moderately radio-sensitive. (J. D.)

Tolerance to Ethyl Alcohol.—Using dogs, it was shown that the animals acquired a definite tolerance to moderate concentrations of blood alcohol. This acquired tolerance to alcohol is not explicable by increased oxidation nor by delayed absorption. (J. D.)

Vol. 86. No. 5. November 1937.

History of Psychiatry and Mental Hospitals in Mexico. S. R. Moreno. 513.

The Rorschach Inkblot Method in Organic Disturbances of the Central Nervous System. Z. Piotrowski. 525.

The Psychodynamics of Chronic Alcoholism. R. P. Knight. 538.

Rosenthal Fibers in Non-Neoplastic Syringomyelia. A. F. Liber and J. R. Lisa. 549.

The Effect of Ergotamine Tartrate in Idiopathic Epilepsy. A. E. Loscalzo. 559.

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Personality Studies in Alcoholic Women. F. J. Curran. 645.

Changes in the Brain in Accidental Electrocution. G. B. Hassin. 668.

The Use of Graphology in Medicine. G. C. Booth. 674.

*Recurrent Trigeminal Neuralgia (Migrainous) associated with Cyclic Scotomata. C. W. Lippman. 680.

Experimental Induction of Infantile Behavior in Major Hysteria. R. R. Dieterle and E. J. Koch. 688.

Recurrent Trigeminal Neuralgia.—A report of a group of cases of migraine in women with pain in the maxillary and mandibular divisions. The attacks were usually related to the menstrual cycle and were associated with enlargement of the blind spot, diplopia, and œdema of the face. Estrin alleviated the condition. (J. D.)

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Vol. 7. No. 1. 1938.

Histopathology of Brain in Methyl-Alcohol Poisoning. A. J. Geimanovic and M. Haet. 3.

The Nosological Place of Scapuloperoneal Amyotrophy. S. Davidenkov and E. Kulkova. 11.

A Rational Classification of the Architectonic Formations of the Brain Cortex based on the Study of Ontogenesis. J. N. Filimonov. 21.

The Clinical Picture of Neurobrucellosis. A. V. Triumfov. 28.

Early Headaches in Brain Tumours. M. Neiding. 38.

Disturbances of Sensibility in Funicular Myelosis. Z. L. Lourie. 42.

Optico-Encephalomyelitis. D. S. Futer. 50.

Post-vaccinal Encephalomyelitis. D. A. Melzer. 60.

Sensory Disturbances in Syringomyelia. I. F. Hvilivizkaia. 69.

Clinical Picture and Patho-physiology of Protective Reflexes of Upper Extremities in Cerebral Affections. S. J. Rotenberg. 79.

Remote Reactions of the Nervous System in Response to Electric Trauma. L. J. Aleksandrova. 94.

Diffuse Melanoma of Soft Meninges of the Brain and Spinal Cord. J. S. Sergeeva. 102.

Reactions to Histidine in Brain Tumours and the Rôle of Histamine in Pathogenesis of Tumours. M. A. Hazanov. 107.

Mortality from Pneumography in Verified Brain Tumours. M. D. Ryndin and L. J. Blachman. 117.

Imbecility in Schizophrenia. A. A. Černuha. 125.

The Prognostic Value of Acute Outset in Schizophrenia. A. D. Grin. 145.

On the So-Called "Psychopathoid" States in Pre-School Children. V. P. Kudriavzeva. 153.

Bourgignon's Ionogalvanization in Atrophy of Ocular Nerve. V. A. Lukásev. 164.

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The Potassium and Water Contents of Cat Nerves as Affected by Stimulation. W. O. Fenn. 1.

The Effect of Stimulation on the Potassium Content of Limulus Leg Nerves. A. C. Young. 4.

The Non-Centrifugal Degeneration of Severed Peripheral Nerve. O. Sugar. 7.

An Oscillographic Study of the Cerebello-Cerebral Relationships. A. E. Walker. 16.

*Human Brain Potentials during the Onset of Sleep. H. Davies, P. A. Davies, A. L. Loombs, E. N. Harvey, and G. Hobart. 24.

The Behavior of Cats following Bilateral Removal of the Rostral Portion of the Cerebral Hemispheres. H. W. Magoun and S. W. Ranson. 39.

Forced Circling Movements in Monkeys following Lesions of the Frontal Lobe. M. A. Kennard and L. Ectors. 45.

*The Representation of Respiratory Movements in the Cerebral Cortex. W. K. Smith. 55.

Functional Organization in the Sensory Cortex of the Monkey (*Macacca mulatta*). J. G. Dusser de Barenne and W. S. McCulloch. 69.

Human Brain Potentials during Sleep Onset.—Electro-encephalographic studies have been carried out on subjects while they were going to sleep. Subjects with good alpha-wave rhythms showed a depression of the alpha waves and the appearance of delta waves as they fell asleep. During sleep slow waves predominated.

The authors point out the necessity to avoid the taking of action potentials when the subject is in a drowsy state. (E. A. C.)

Respiratory Movements in Cerebral Cortex.—It has been possible to produce alterations in the respiratory rhythm by electrical stimulation of the cerebral cortex in cat, dog, and monkey. Both inhibition and acceleration have been obtained. Automatic somatic responses have also been obtained, but these have been excluded as causes of the alteration in respiratory rhythm. (E. A. C.)

Vol. 1. No. 2. March 1938.

*Brain Potentials and Voluntary Muscle Activity in Man. H. H. Jasper and H. L. Andrews. 87.
*Activation of Heat Loss by Local Heating of the Brain. H. W. Magoun, F. Harrison, J. R. Brobeck, and S. W. Ranson. 101.
*Localized Thermal Changes in the Cat's Brain. H. M. Serota and R. W. Gerard. 115.
On an Ipsilateral Motor Effect from Cortical Stimulation in the Macaque Monkey. O. A. M. Wyss. 125.
On the Kinetics of Recovery during the Refractory Period in Frog's Nerve. H. A. Blair. 127.
Functional Recovery in Ocular Muscles of a Chimpanzee after Section of Oculomotor Nerve. M. B. Bender and J. F. Fulton. 144.
*Electrical Excitability of the Motor Face Area : a Comparative Study in Primates. A. E. Walker and H. D. Green. 152.
Facilitation of Reflex Action in the Spinal Cat following Asphyxia of the Cord from Cyanide and Increased Intra-Spinal Pressure. E. L. Porter, R. K. Blair, and S. W. Bohmfalk. 166.
The Direct Functional Interrelation of Sensory Cortex and Optic Thalamus. J. G. Dusser de Barenne and W. S. McCulloch. 176.

Brain Potentials and Muscle Activity.—Muscle potentials, tremor, and brain potentials have been recorded simultaneously. Tremor movements of the fingers in normal subjects were studied ; the average frequency of the tremor was about 10 per second. Such frequencies correspond with the alpha and beta rhythms of cortical potentials. The authors also studied two cases of unilateral paralysis agitans. The abnormal tremor of 4 to 5 per second in these cases did not appear in the cortical potential rhythm. (E. A. C.)

Heat Loss by Local Heating of Brain.—In the medial portion of the caudal part of the ventral telencephalon of cats are elements which react to local heating. This results in increase in respiratory rate, panting, and occasionally sweating. (E. A. C.)

Thermal Changes in Cat's Brain.—From a study with thermojunctions placed in the cat's brain, it has been found that there is a temperature gradient from the surface to the interior. Stimulation of visual function caused changes in the visual system of the brain, and of olfactory function changes in the olfactory regions. They suggest that the extra heat produced results from the activity of neurones and their metabolic products. (E. A. C.)

Electrical Excitability of Motor Face Area.—The authors found from their work on the monkey, baboon, and chimpanzee that movement of the tongue is bilaterally represented in the cerebral cortex and that movements of the vocal cords may be obtained from Area 6. (E. A. C.)

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Vol. 91. No. 1. October 1937.

The Discharge of Impulses from Ganglion Cells. J. C. Eccles. 1.
The Energy Liberation at Constant Diastolic Fibre Length in the Tortoise Heart with Particular Reference to the Effect of the Emptying Pressure. L. F. Moldavsky and M. B. Visscher. 23.
A Simple Direct-Coupled Amplifier for Action Potentials. A. Forbes and A. M. Grass. 31.
The Sensation of Yellow and Anomalous Trichromatism. J. G. Hailwood and H. E. Roaf. 36.
The Influence of the Reaction of Blood Plasma on Oxygen Consumption in Relation to the Law of Isodynamic Equivalence. F. Kane and J. M. O'Connor. 48.
The Relation between Respiratory Quotient and Alveolar CO_2 Tension. O. Fitzgerald and J. M. O'Connor. 59.
Synchronized Reactions in the Optic Ganglion of *Dytiscus*. E. D. Adrian. 66.
*The Relation between Plasma and Cerebrospinal Fluid Calcium. A. T. Cameron and V. H. K. Moorhouse. 90.

Calcium in Plasma and C.S.F.—From a series of experiments in dogs it was found that the ratio of cerebrospinal fluid calcium to serum calcium averaged 50 per cent. They concluded from further experiments with injection of calcium salts or of parathormone that the cerebrospinal fluid calcium represents the diffusible fraction of the plasma calcium. (E. A. C.)

Vol. 91. No. 2. November 1937.

The Effects of Tetra-ethyl-ammonium Iodide on the Electrical Response and the Accommodation of Nerve. S. L. Cowan and W. G. Walter. 101.
Excitation and Inhibition in the Off-Effect of the Retina. R. Granit and P. O. Therman. 127.
The Origin and Limits of the Specific Dynamic Action of Intravenous Glucose. M. Wierzuchowski. 140.
The Relation between the Mechanical and Electrical Activity of a Molluscan Unstriated Muscle. C. M. Fletcher. 172.
*The Cerebrospinal Fluid Pressure of Man in the

Erect Posture. E. A. Carmichael, J. Doupe, and D. J. Williams. 186.
The Pituitary Gland and the Control of Urinary Secretion. F. C. Dodds, R. L. Noble, and P. C. Williams. 202.
*The Local Action of Eserine on the Central Nervous System. F. R. Miller. 212.
*The Secretion of Urine in Man during Experimental Salt Deficiency. R. A. McCance and E. M. Widdowson. 222.

C.S.F. Pressure of Man in Erect Posture.—A study has been made of the cerebro-spinal fluid pressure in the erect posture of man. Evidence is produced to show that in the erect posture fluctuations of cerebro-spinal fluid pressure follow changes in blood pressure. They also draw attention to the venous pressure in the jugular bulb and how the pressure remains unaltered during respiration or during deep inspiration. No evidence is obtained that vaso-constriction of the intracranial vessels occurred on electrical stimulation of the stellate ganglia. (E. A. C.)

Action of Eserine on Central Nervous System.—Application of eserine sulphate to the motor cortex under dial anæsthesia gives rise to tremors, muscular rigidity, and clonus in the contralateral limbs. It is suggested that eserine causes a local facilitation. (E. A. C.)

Urine Secretion in Salt Deficiency.—Salt deprivation and marked sweating in man was accompanied by a rise in blood urea. It is suggested that the salt deficiency led to a diminished glandular filtration. (E. A. C.)

Vol. 91. No. 3. December 1937.

Observations on the Inactivation of Adrenaline by Blood and Tissues *in vitro*. W. A. Bain, W. E. Gaunt, and S. F. Suffolk. 233.
The Mechanism of Pitch Change in the Voice. R. Curry. 254.
The Effect of Anaerobic Activity and Rest on the Efficiency of Contraction in Isolated Frog Muscle. McK. Cattell and H. Feit. 259.
The Red Cell Count of Macaques in Relation to the Menstrual Cycle. A. N. Guthkelch and S. Zuckerman. 269.
The Acclimitization of Mice to Atmospheres containing Low Concentrations of Carbon Monoxide. E. M. Killick. 279.
The "After-Positivity" of Stimulated Nerve and its Relation to the First Phase of the Recovery Heat Production. J. L. Parkinson. 293.
The Transmission of Impulses through the Inferior Mesenteric Ganglia. D. P. C. Lloyd. 296.
Changes in Creatine, Phosphagen, and Adenylpyrophosphate in Relation to the Gaseous Metabolism of the Heart. W. Burns and E. W. H. Cruikshank. 314.
Movements in Midfœtal Life in the Sheep Embryo. J. Barcroft and D. H. Barron. 329.
The "Pancreotropic" Action of Anterior Pituitary Extracts. K. C. Richardson and F. G. Young. 352.

Vol. 91. No. 4. January 1938.

Effects of Castration and of Sexual Hormones on the Adrenals of Male Rats. K. Hall and V. Korenchevsky. 365.
The Response of a Denervated Ganglion to Acetylcholine. F. T. v. Brücke. 375.
Lipoid Substances of the Ovary during Ova Production in *Rana Pipiens*. E. M. Boyd. 394.
Factors affecting the Sodium, Potassium, and Tatal Base Content of the Anterior Retractor of the Byssus of *Mytilus Edulis*. I. Singh. 398.
Exclusion of the Liver in the Rabbit. H. P. Himsworth. 413.
Binocular Summation during Dark Adaptation. R. J. Lithgoe and L. R. Phillips. 427.
The Action of Curarine on the Respiratory Mechanism. R. West. 437.
The Relation of the Hypophysis to Changes in Sugar Tolerance and Insulin Sensitivity induced by Changes of Diet. H. P. Himsworth and D. B. McN. Scott. 447.
The Action of Tyramine and Adrenaline on the Denarvated Nictitating Membrane. E. Bülbring and J. H. Burn. 459.
The Carbonic Anhydrase Inhibitor in Serum. V. H. Booth. 474.

Vol. 92. No. 1. February 1938.

Response of the Cat's Uterus to the Hormones of the Posterior Lobe. J. M. Robson and H. O. Schild. 1.
Effect of Drugs on Blood Flow and Activity of the Uterus. J. M. Robson and H. O. Schild. 9.
Heparin and the Formation of White Thrombi. C. H. Best, Cowan, Campbell, and D. L. Maclean. 20.
A Note on the Effects of Vitamin-A Deficiency in Pregnant Rats. W. H. Newton. 32.
Choline Esterase in Voluntary Muscle. A. Marnay and D. Nachmansohn. 37.
On the Behaviour of Glycogen after Diets Rich in Protein and in Carbohydrate. A. Mirski, I. Rosenbaum, L. Stein, and E. Wertheimer. 48.
The Effect of the Interaction of Ions, Drugs, and Electrical Stimulation as indicated by Contraction of the Anterior Retractor of the Byssus of *Mytilus edulis*. I. Singh. 62.
The Effect of Anterior Pituitary Extracts on the Liver Fat of Various Animals. C. H. Best and J. Campbell. 91.
The Central Depressor Action of Adrenaline and Its Inhibition by Ergotoxine. U. S. v. Euler. 111.
The Effect of Hypophysectomy on the Blood Calcium and Phosphorus of the Rat. A. B. Anderson and E. G. Oastler. 124.

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The Effect of Certain Hormones on the Activity of the Uterine Muscle of the Mouse. G. H. Bell and J. M. Robson. 131.
The Effect of the Placenta on the Body Weight of the Mouse. J. B. Brooksby and W. H. Newton. 136.
The Influence of Hydrogen-Ion Concentration on the Surface Tension of Some Colloidal Solutions. P. W. Perryman and C. F. Selous. 151.
*Sensory Fibres in the Spinal Accessory Nerve. F. Echlin and N. Propper. 160.
Observations in Man on a Pulse-Accelerating Reflex from the Voluntary Muscles of the Legs. M. Alam and F. H. Smirk. 167.

The Action of Creatine and Creatinine on the Circulatory System. B. G. Shapiro. 178.
The Action of Young's Glycotropic Factor of the Anterior Pituitary Gland. H. P. Himsworth and D. B. McN. Scott. 183.
*The Effect of Salt Deficiency in Man on the Volume of the Extracellular Fluids, and on the Composition of Sweat, Saliva, Gastric Juice, and Cerebrospinal Fluid. R. A. McCance. 208.
The Carbohydrate Metabolism of the Fœtal Dog under the Influence of Insulin. H. Schlossmann. 219.

Sensory Fibres in Spinal Accessory Nerve.—A study was made in cats of the action potentials obtained from the spinal accessory nerve. The authors concluded that they came from stretch receptors in the muscles supplied by the nerve. (E. A. C.)

Salt Deficiency and Extracellular Fluids.—During experimental production of salt deficiency the C.S.F. was examined in two subjects. It was found that the sodium content of the C.S.F. had fallen and that the concentration of chlorides had fallen to about 640 mgm. per cent. However, the chlorides in the plasma had fallen to a lower level. (E. A. C.)

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Spectrophotometer Investigation into the Differences between Fœtal and Maternal Hæmoglobin in Man. J. Jongbloed. 229.
Isotonic Extension and Shortening of the Anterior Retractor of the Byssus of *Mytilus edulis*. I. Singh. 232.
The Effect of Adaptation to Electrical and Chemical Stimulation of the Excitability of the Anterior Retractor of the Byssus of *Mytilus edulis*. I. Singh. 241.
Some Conditions of Fœtal Respiration in the Cow. J. Roos and C. Romijn. 249.
The Electrocardiogram of the Guinea-Pig. C. L. G. Pratt. 268.
Antibodies Organ-Specific against the Anterior Body of the Pituitary Gland. O. Kestner. 273.
The Interpretation of Potential Changes in the Spinal Cord. D. H. Barron, B. H. C. Matthews. 276.
The Action of Narcotics on Brain Respiration. M. Jowett. 322.
The Buffering of Muscle in Rigor: Protein, Phosphate, and Carnosine. E. C. Bate Smith. 336.
Blood Histamine and Cardiac Activity. C. F. Code, C. Lovatt Evans, and R. A. Gregory. 344.
The Osmotic Pressure of the Aqueous Humour in the Normal and Glaucomatous Eye. G. H. Benham, W. S. Duke-Elder, and T. H. Hodgson. 355.

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Vol. 48. Nos. 1 and 2. 1938.

Zur Normalanatomie der Substantia nigra. Versuch einer architektonischen Gliederung. (Cytarchitecture of the substantia nigra.) R. Hassler. 1.
Comparative Anatomy of the Regio Entorhinalis and Regio Præsubicularis. K. Sgogina. 56.

PHYSIOLOGICAL REVIEWS

Vol. 18. No. 1. January 1938.

The Relation of the Anterior Pituitary to Carbohydrate Metabolism. J. A. Russell. 1.
Physiology of Excretion among the Anthropoda. N. S. Royston Maloeuf. 28.
Recent Advances in Knowledge concerning Hearing and Speech. P. M. Tookey Kerridge. 59.
Arterial and Venous Pressure Factors in Circulatory Failure. T. R. Harrison. 86.
The Nutritive Significance of the Amino Acids. W. C. Rose. 109.

Vol. 18. No. 2. April 1938.

Sympathetic Vasodilator Fibres. J. H. Burn. 137.
The Sites of Formation of Estrogenic Substances in the Animal Body. G. W. Corner. 154.
Autoclysis and Atrophy. H. C. Bradley. 173.
The Causes and Mechanism of Thrombosis. M. Silberberg. 197.
Arterio-Venous Anastomoses. E. R. Clark. 229.
Undernutrition and Carbohydrate Metabolism. W. H. Chambers. 248.
Present Status of the Ergot Question. E. E. Nelson and H. O. Calvery. 297.

PSYCHIATRIC QUARTERLY

Vol. 12. No. 1. January 1938.

*Some Observations in the Treatment of Dementia Præcox with Hypoglycemia. M. Schatner and F. J. O'Neill. 5.
A Study of the Impairment of "Abstract Behavior" in Schizophrenic Patients. M. Bolles and K. Goldstein. 42.
*The Clinical Effect of Benzedrine Sulphate in Mental Patients with Retarded Activity. L. F. Woolley. 66.
*Serology in General Paresis. J. L. Blalock and L. E. Hinsie. 84.

Intraventricular Meningiomas. A. Ferraro and J. Siris. 117.
The Rôle of Physical Therapy in the Treatment of Nervous and Mental Diseases. R. Kovacs. 123.
Psychiatric Aspect of Head Injuries. I. N. Wolfson. 137.
Technical Approaches used in the Study and Treatment of Emotional Problems in Children. J. L. Despert. 176.

Treatment of Dementia Præcox with Hypoglycæmia.—Seventy-five cases were treated with hypoglycæmia. The treatment in 56 of these cases has been completed, and the remainder are still under treatment. The authors emphasize the following points. The marked variation in the amount of insulin required to produce coma, the danger of aspiration pneumonia, and the substantial gain in weight in the patients regardless of the degree of mental improvement shown. Of the 56 completed cases 57 per cent. (=32) showed a definite benefit from the treatment. The authors give no information or tables that could assist in assessing the relation of the efficacy of their treatment to the duration of the illness.

Clinical Effects of Benzedrine Sulphate.—A total of 70 cases was collected ; 44 were diagnosed as schizophrenic ; 21 as manic depressive ; 25 as psychoneurotic. No patients over 50 or with high blood pressure were included in this group. These cases were treated with benzedrine sulphate over a period of some 6–9 months. It could be demonstrated that about 25 per cent. of the retarded patients in all the diagnostic groups could be improved by administration of the drug. About 25 per cent. of the retarded patients in all diagnostic groups were made much worse by its influence. Approximately 50 per cent. appeared to be unaffected. The response to benzedrine could not be predicted from the clinical examination of the patients nor from the application of pharmaco-diagnostic tests. In general those patients who showed evidence of fear and anxiety or agitation were either not materially affected by benzedrine sulphate or made worse. Patients who reacted favourably to the treatment showed almost immediate improvement on the administration of 10-mg. doses over a period of from one to three days.

Serology in General Paresis.—The data presented in this paper cover all the available serological information on patients treated at the New York State Psychiatric Institute and Hospital from 1932–1935 inclusive. During this period there were over 300 patients under treatment. The cases analysed were treated by malaria, tryparsamide, and electropyrexia. During the early months of tryparsamide therapy the blood Wassermann became negative in a far greater proportion than it did in those patients treated by other methods. At the end of three months 40 per cent. of patients treated by tryparsamide had a negative blood W.R., whereas the percentage was 16·1 with malaria, 9·4 with electropyrexia and chemotherapy, and 6·4 with electropyrexia alone. At the end of six months, tryparsamide therapy still maintained its lead. At the end of three years the percentage of negative W.R. in the blood group treated by electropyrexia and tryparsamide alone was 40·0 per cent., and 29·5 per cent. with malaria therapy, 18·7 per cent. under electropyrexia alone, and 16·2 per cent. under tryparsamide alone. At the end of seven years the percentage of negative blood W.R. from the malarial group was 68·4 whilst it was 58·3 in those treated with tryparsamide alone. No single method of treatment gave such good results as a combination of fever and chemotherapy. The spinal fluid W.R. was at the end of the first year negative in 28·9 per cent. of patients treated by electropyrexia and tryparsamide. For tryparsamide alone the figure was 25·5 per cent., electropyrexia alone 15·6 per cent., and malaria alone 5·2 per cent. At the end of three years the percentages were 42·1 per cent., 27·8 per cent., 18·7 per cent., and 40·5 per cent. respectively. After a period of seven years the percentage of the negative spinal fluid W.R. was essentially the same ; namely, 85·7 per cent., and 90 per cent. under malarial treatment alone and tryparsamide treatment alone. The authors agree with the majority of investigators in pointing out that serological improvement is in general independent of the clinical condition of the patient, but add that a persistently positive serological ensemble is usually associated with an unfavourable clinical condition.

Vol. 12. No. 2. April 1938.

Review of the Research Efforts of the New York State Psychiatric Institution and Hospital during the Year 1937. N. D. C. Lewis. 217.
*Aspects of the Electroencephalogram in Epilepsy and Feeblemindedness. W. E. Rahm and A. C. Williams. 230.
Is the Paroled Patient a Menace to the Community ? H. M. Pollock. 236.
*The Use of Methylene Blue in the Treatment of Catatonic Dementia Præcox Patients. W. J. Allexsaht. 245.
Concerning Hallucinations of Smell. G. M. Davidson. 253.
Psychological Elements in Continuous Bath Therapy. C. N. Allen. 271.
Sudden "Exhaustive" Death in Excited Patients. N. R. Shulack. 282.
Pathological Changes in the Brain in Cases of Experimental Phosphorus Intoxication. A. Ferraro, G. A. Jervis, and W. H. English. 294.
A Study of the Problem of Suicide. B. Pollack. 306.
Folie à Deux. J. A. Brussel. 331.
Hyperostosis Frontalis Interna. O. C. Perkins and A. M. Biglan. 341.
Psychotherapy in Neurotic Patients with Somatic Disease. E. Davidoff. 351.
Schizophrenia in Children. J. L. Despert. 366.
*The Epileptic Seizure in the Hypoglycemic Treatment of Schizophrenia. D. Ruslander. 372.
The Startle Pattern in Epileptic Patients. W. A. Hunt, H. Strauss, and C. Landis. 375.

Electroencephalogram in Epilepsy.—Records were obtained from 35 unselected feeble-minded patients with a mental age range from less than one year to 9·8 years, and for 48 unselected epileptic patients. After elimination of records where movements predominated, the final groups considered numbered 27 feeble-minded and 35 epileptic patients. The distribution of alpha wave activity was plotted for each group. In studying the alpha wave content of the records the " per cent. time alpha " was used as a measure. This was determined by dividing the time taken up by alpha activity by the total time of recording and multiplying this result by 100. In the feeble-minded group a negative correlation (—0·54—0·09) was obtained between the percentage time alpha of the electroencephalogram and the chronological age. In the epileptic group no correlation (—0·04—0·10) was obtained between the per cent. time alpha of the electroencephalogram and the chronological age.

Methylene Blue in Treatment of Catatonic Dementia Præcox.—Five cases of dementia præcox with catatonia were treated with intravenous injections of zinc-free methylene blue. A 1 per cent. methylene blue solution was made up in double distilled water and autoclaved at 15 lb. No harmful effects were noted from the injection of 50 c.c. of the solution which was given slowly taking about 5 minutes to complete the injection. Only a single injection was given to each case. The accounts of the results of this treatment do not afford convincing evidence of any therapeutic action.

Hypoglycæmic Treatment of Schizophrenia.—In a series of 67 cases treated at the Buffalo State Hospital from 8th March, 1937 to 6th February, 1938, 18 cases were observed to have experienced typical epileptic seizures from 1–9 times during the treatment ; they constitute 27 per cent. of the entire group. Twelve patients appeared to have been benefited by such seizures and were able to return to their homes, where they continue to make good progress. In three cases improvement was noted immediately following occurrence of a seizure. The authors consider that the occurrence of epileptic seizures is not only without danger to the patient but in some instances a decisive factor in bringing about improvement.

REVUE NEUROLOGIQUE

Vol. 68. No. 4. October 1937.

Contribution à l'Étude des Troubles Sensitifs Hystériques et le Rôle des Réflexes Conditionnels dans la Physio-Pathologie de l'Hystérie. (Contribution to the study of hysterical disturbances of sensation.) G. Marinesco. 585.

Étude Expérimentale des Mouvements Involontaires. (Experimental study of involuntary movements.) N. Zand. 601.

Hémihyperpathie du Goût. (Hemihypertrophy of taste.) I. Silberpfennig and H. Urban. 613.

Vol. 68. No. 5. November 1937.

*Les Hématomes de la Dure-Mère, Diagnostic, Pathogénie, Traitement. (Étude Portant sur 34 Cas Vérifiés.) (Hematoma of the dura mater—diagnosis, pathogenesis, treatment. A study of 34 verified cases.) G. de Morsier. 665.

Hematomas of the Dura Mater.—An extensive survey of the literature on subdural hematoma with a report of 34 verified cases. The author favours the view that the hematoma develops in the layers of the dura. (E. A. C.)

Vol. 68. No. 6. December 1937.

*Pinéalome avec Métastases Multiples. (Dissémination par le Liquide Céphalo-Rachidien.) (Multiple Metastases in Pinealoma. Dissemination of the Cerebrospinal Fluid.) C. Alajouanine, C. Hornet, and R. Thurel. 793.

*Sur une Forme à Évolution Phasique de la Leuco-encéphalose Périvasculaire Confluente chez le macacus Rhésus. (Type Cérébral Répondant à la Myélose Funiculaire du Singe.) (Leuco-encephalitis in monkeys.) H. J. Scherer. 807.

Multiple Metastases in Pinealoma.—A report of a case of pinealoma with secondary deposits in the lateral and third ventricles and meninges of the spinal cord. (E. A. C.)

Perivascular Leuco-encephalitis in a Monkey.—The author describes a case of perivascular leuco-encephalitis in a monkey. Unfortunately the spinal cord was not examined. During life the monkey showed no symptoms. (E. A. C.)

Vol. 69. No. 1. January 1938.

Sur l'Idiotie Amaurotique Juvénile (Type Spielmeyer-Vogt). Contribution à l'Étude de sa Sémiologie Extrapyramidale, de la Répartition et de la Cytologie de ses Lésions. (Juvenile amaurotic idiocy (Spielmeyer-Vogt type). M. Dide and L. van Bogaert. 1.

Vol. 69. No. 2. February 1938.

Syndromes Céphalgique et Psychoévrotique avec Calcifications de la Faux du Cerveau. (Cerebral symptoms associated with calcification of the falx.) J. A. Chavany and J. Brunhes. 113.

Vol. 69. No. 3. March 1938.

*Étude Anatomo-Clinique sur une Lésion Ancienne du Noyau Rouge. (Anatomo-clinical study of an old lesion of the red nucleus.) G. Guillain, I. Bertrand, and J. Guillain. 233.

Technique de Mise en Évidence des Gaines de Myéline des Fibres Nerveuses sur Coupes Histologiques à la Paraffine. (Myelinated fibres. Histological sections in paraffin.) Y. Poursines. 248.

Lesion of Red Nucleus.—A case is described in which an old lesion of part of the red nucleus was found. The degenerations found elsewhere, such as in the olive, are discussed in relation to the lesion in the red nucleus. (E. A. C.)

Vol. 69. No. 4. April 1938.

Les Méningites Séreuses. (Serous meningitis.) M. Molhant. 329.

Nouvelle Observation Anatomo-Clinique de Sclérôse en Plaques Aiguë. (A new observation on disseminated sclerosis.) Riser and Geraud. 248.

RIVISTA DI NEUROLOGIA, NAPOLI

Vol. 10. No. 6. December 1937.

*Sulla Neuromielite Ottica. (On neuromyelitis of the optic nerve.) M. Gozzano. 529.

*Comportamento della Fibra Nervosa di Fronte All'Azione della Corrente Galvanica e della Corrente Faradica (Behaviours of nerve fibres exposed to the action of galvanic and faradic currents.) G. Oggioni. 574.

*Contributo allo Studio Istologico dei Gangli Spinali. (Contribution to the histological study of the spinal ganglia.) V. della Pietro. 588.

Neuromyelitis of the Optic Nerve.—A study of a case which forms the basis of a discussion of the true nature of optic neuromyelitis. The author thinks that in view of the clinical and histopathological characteristics the condition ought to be regarded as quite distinct from all other non-suppurative affections of the neuraxis whether diffuse or disseminated. On the clinical side it is characterized by the coexistence of a syndrome of transverse myelitis and optic neuritis and on the anatomical side by the absence of a neuroglial repair reaction and by the practically constant tendency to necrosis. (R. G. G.)

Nerve Fibres exposed to Galvanic and Faradic Currents.—Using the method of Donaggi's the effects of currents of varied duration on nerve fibres were observed. Minute modifications were shown consisting to begin with of increased absorption of stain by the fine structure of the myeline in electrolysed nerves. With more intense application of current the axis and myeline show a homogeneous mass of stain with a type of primary initial degeneration. There is a correspondence between the reversible alterations in function induced by the currents and the colour changes observed. (R. G. G.)

Spinal Ganglia.—The structure and appearance of the various cells are described and the identity of the pericellular elements and oligodendroglia demonstrated. (R. G. G.)

Vol. 11. No. 1. February 1938.

*Recherches Anatomo-Cliniques sur les Polyradiculo-Nevrites et leurs Rapports avec les Poly-Névro-Radiculo-Myélites. (Anatomico-clinical investigation of the forms of polyradiculo-neuritis and their correspondence with forms of polyradiculo-myelitis.) G. Marinesco. 1.

*L'Azione Combinata dell'Armina e della Bulbocapnina sui Gatti. (The combined action of armine and bulbo-capnine in cats.) P. Penta and A. Aveta. 31.

*Sulla Riattivazione della R. di Wassermann nel Liquido Cefalo-Rachidiano. (The reactivation of the Wassermann reaction in the cerebrospinal fluid.) V. Della Pietra. 55.

*Equivalenti Psichici Atipici in Seguito a Trauma Cranico. (Atypical psychic equivalents following cranial trauma.) G. Glück. 71.

Polyradiculo-Neuritis and Polyradiculo-Myelitis.—The syndrome of Guillain and Barré — Polyradiculo-neuritis — first described in 1916, has been confirmed by many observers, and it has been suggested that the same condition may affect the spinal cord and more rarely the bulb and the brain. The forms which show excess of albumin in the C.S.F. but not increase in cells show two clinical forms, the curable syndrome of Guillain and Barré and the fatal form which is just like the first, but affecting as it does the respiratory nerves has a fatal issue. The post-mortem findings of two cases are described, which differed inasmuch as in one nerve roots and fibres only were affected while in the other both white and grey matter of the cord showed marked inflammatory changes. It would seem that these conditions are both due to an ultra virus different from the neurotrophic virus of poliomyelitis rabes, etc., which produces inflammatory reactions which may be reversible. (R. G. G.)

Action of Armine and Bulbocapnine in Cats.—It is evident that distinct phenomena are observable; the abolition of

catatonia and the suppression of the cerebellar function is characteristic of the action of armine and correspondingly onset of catatonia and the lessening of the defect of cerebellar function is produced by bulbocapnine. These phenomena, while showing a pharmacological antagonism between the two drugs, do not mean that there is a cerebellar factor in catatonia, but show the wide field of action of these two alkaloids throughout the neuraxis. (R. G. G.)

Reactivation of Wassermann Reaction in C.S.F.—Experiments on the provocative effect on the Wassermann reaction in the cerebrospinal fluid by the injection of 5 per cent. pheno-sulphothalein do not hold any hope that this procedure is likely to be of practical clinical use. (R. G. G.)

Atypical Psychic Equivalents following Cranial Trauma.—Apparently psychic effect of post-traumatic epilepsy do not resemble the psychic manifestations of idiopathic epilepsy, but rather the general psychic sequelæ of cerebral trauma not associated with epilepsy. In the case recorded by the author, which is exceptional to judge by the literature, the traumatic ætiological factor can be deduced from the clinical nature of the psychic epilepsy. (R. G. G.)

RIVISTA DI PATALOGIA NERVOSA E MENTALE

Vol. 50. Part 3. November 1937.

*Sul Morbo di Marchiafava (Degenerazione Primitiva Sistematizzata delle Vie Commessurali dell'-Encefalo). (On Marchiafava's disease (primary systematized degeneration of the commissural tracts of the brain).) G. Fittipaldi. 427.
*Contributo allo Studio della Patogenesi della Miastenia. (Contribution to the study of the pathogenesis of myasthenia.) C. Trabucchi. 470.
*Parallelismi Umorali tra Epilessia e Stati Allergici. (The parallel condition of the body fluids in epilepsy and allergic states.) S. Segre. 504.
*Studio Anatomo-Patologico di un Caso di Malattia Epato-Lenticolare di Wilson. (An anatomico-pathological study of a case of Wilson's disease.) G. Vidari. 520.
*Le Alterazioni del Reticulo Neurofibrillare Endocellulare nella Fatica Sperimentale. (The alteration in the endocellular neurofibrillary network in experimental fatigue.) G. Oggioni. 548.
*Sindrome Onirica da Malaria (Contributo Clinico con Analisi del Delirio Onirico.) (Hallucinatory syndrome in malaria.) G. de Gregorio. 557.

Marchiafava's Disease.—Forty years ago Marchiafava and Bignami drew attention to a degeneration of the corpus callosum in alcoholics. Since then 46 cases have been described and two further cases are here presented. The author concludes that this disease represents a well-defined anatomical picture consisting of a symmetrical demyelinization with a selective localization in the commissural fibres. Alcoholism is very frequent in patients with the disease, while other agencies, such as malaria, lead poisoning, or diabetes, are correspondingly rare. It would appear therefore that alcohol is an important ætiological factor, but the true pathology of the disease is quite obscure. (R. G. G.)

Pathogenesis of Myasthenia.—The author, in order to establish whether in myasthenia there is a disturbance of the chemical transmission of the neuromuscular impulse allied to the formation of acetylcholine, has subjected a severe case of the disease to a variety of tests, with the following results. The quantity of cholinesterase contained in the blood and in the serum was not increased to any greater degree than in the normal subjects. No disturbances in the vegetative sphere could be discovered, but the innervation of the smooth muscle was not quite normal, as there was a constant lowering of the tone of the stomach wall. Prostigmine in therapeutic doses was found to modify the reaction of the stomach wall and to restore the tone to normal. (R. G. G.)

Body Fluids in Epilepsy and Allergic States.—Modifications of body fluids observed in cases of allergic states, particularly a tendency to increase of fluid in the tissues, increased volume of the red blood corpuscles, and toxicity of the blood serum, were investigated in persons suffering from idiopathic epilepsy. These changes showed a certain variation but were specially marked at the time of attacks, especially if these were frequent. Next the effect of magnesium sulphate was tried in view of the beneficial effects of its administration in allergic states in general. *In vitro* it seemed to have a marked effect in reducing the toxicity of the serum, and administered intravenously up to 25 per cent. of magnesium sulphate caused some improvement in the equilibrium of the body fluids and a sensible reduction in the frequency and intensity of the attacks. (R. G. G.)

Wilson's Disease.—This was a man of 50 who showed cirrhosis of the liver with large nodules, splenomegaly without ascites or jaundice, and almost complete destruction of the putamen, and in addition —a new observation—a complete destruction of the corpus Luysei. Microscopically there appeared a marked proliferation of fibrous glia in the putamen, though they were scarce in the other affected nucleus. Also the presence in various parts of the brain, but especially in the putamen and corpus Luysei, of large collections of glial cells. (R. G. G.)

Alteration in Endocellular Neurofibrillary Network in Experimental Fatigue.—A characteristic appearance was seen when

stained by Donaggio's method of agglutinization which varied in different parts, the anterior horn cells seeming to be particularly resistant. (R. G. G.)

Hallucinatory Syndrome in Malaria.—A case is described and discussed and the relationship of the hallucinations and confusion. The author thinks that the psychoses of malaria are due to both mechanical and toxic factors (the presence of parasites and the poison of the plasmodia). (R. G. G.)

Vol. 51. No. 1. January 1938.

*Contributo alla Conoscenza dell' Epilessia Sperimentale Riflessa per Stimoli Luminosi. (A contribution to the knowledge of experimental reflex epilepsy induced by luminous stimuli.) E. Fulchignoni. 1.

*Due Casi di Cisticercosi Cerebrale Diagnosticati in Vita. (Two cases of cerebral cystocercosis diagnosed during life.) G. Canziani and A Nobile. 55.

*Sull'Uso della Cebtrifugazione nella Reazione del Benzoini Colloidale. (On the use of the centrifuge in the carrying out of the colloidal benzoin reaction.) R. Bozzi. 85.

*Sindrome di Poliomielite Anteriore Acuta nella Sifilide Midollare. (Syndrome of acute anterior poliomyelitis in medullary syphilis.) E. Fenga. 92.

*Sul Tipo " Macrobioticus Multiparus " del Tremore Essenziale. (On the type of hereditary tremor described as Macrobioticus multiparus.) G. Pintus. 114.

*Rilievi Sopra un Nuovo Metodo per lo Studio della Rete Vasale del Sistema Nervosa in Condizioni Normali e Patologiche. (Observations on a new method for the study of the vascular network in the nervous system in normal and pathological states.) C. Fazio. 125.

*I Meningiomi della Fossa Cranica Anteriore ed i Loro Rapporti con la Linea Mediana. Contributo alla Precisazione del Valore Diagnostico dei Sintomi di Lateralita. (The meningiomata of the anterior cranial fossa and their correspondence with the median line. A contribution to the study as to how far the determination of the side of the lesion is of precise diagnostic value.) P. Ottonello. 137.

Experimental Reflex Epilepsy.—The author has investigated by means of various experimental methods the limits of analogy between the phenomena of epilepsy induced by means of the application of strychnine to the occipital area (studied by Clementi) and that of the so-called reflex epilepsy induced by afferent excitement by means of luminous stimuli (studied by Amantea), and comes to the conclusion that whatever analogy there may be really only consists in the occurrence of the epileptic attack.

In both cases the attack results from a spread of impulse through the sensori-motor paths which are normally concerned with the epileptic attack, and that this would not take place were it not for some facilitation of the paths in both cases. (R. G. G.)

Cerebral Cystocercus diagnosed during Life.—The authors, after a discussion of the principal findings in respect of cerebral cystocerci referred to in the literature, lay stress on certain controversial points. They describe two cases in which the diagnosis was made during life by the discovery of a marked eosinophilia in the cerebrospinal fluid. They insist that if a diagnosis is to be made with any confidence the percentage of granular eosinophils should not be less than 1 per cent. The value of this is great, but is perhaps somewhat lessened by the fact that rarely cerebral cystocercus may be met with without changes in the fluid, while very exceptionally eosinophilia may be met with in the cerebrospinal fluid in cases of cerebral syphilis. (R. G. G.)

The Centrifuge in Colloidal Benzoin Reaction.—The author shows that by using the centrifuge the time taken by the test is reduced from hours to minutes and shows by means of tables both of normal cerebrospinal fluids and of pathological fluids how closely the centrifuge method corresponds to the old method of precipitation. (R. G. G.)

Poliomyelitis in Medullary Syphilis.—A case of poliomyelitis is described with peculiar findings in the cerebrospinal fluid and the author after discussing certain similar cases from the literature gives his reasons for supposing that the patient under discussion was suffering from a syphilitic form of acute anterior poliomyelitis. (R. G. G.)

Hereditary Tremor.—In 1925 Minor described 44 families suffering from hereditary tremor with remarkable longevity, and remarked that such families were notably prolific. From his statistical studies, however, the author concludes that the affected families are not more prolific than the average of the population and that the expectation of life is rather less than the average. (R. G. G.)

Vascular Network in the Nervous System.—Pickworth's method here described is based on the benzidine test for blood in urine, and this is applied to sections of the organ to be examined. While not nearly so complete as the method of injecting the vessels with coloured material, it is so quick and simple that it may often be of great advantage when no great detail is desired. (R. G. G.)

Meningiomata of the Anterior Cranial Fossa.—These tumours are of special importance since they lie outside the cerebral tissues, exerting their effects by pressure and being essentially amenable to surgical interference. The occurrence of a bilateral malignant olfactory meningioma which clinically appeared to be situated in the left half of the anterior cranial fossa gives the author an opportunity to discuss some aspects of the diagnostic problem. The author stresses the importance of

tremor, of alterations in the fundus, of the radiographical findings, and above all of the most careful study of the encephalograms in relation to the anatomical land marks in the individual case, since encephalograms may on account of accidental circumstances be very difficult to interpret. In the case of the meningiomata of the anterior fossa it may be very difficult to determine whether the tumour is strictly unilateral or whether it may be implanted on the middle line, and this should be kept in mind in determining the technique of surgical approach. (R. G. G.)

SCHWEIZER ARCHIV FÜR NEUROLOGIE UND PSYCHIATRIE

Vol. 39. (Supplement.) 1937.

*Die Therapie der Schizophrenie. Insulinschock, Cardiazol, Dauerschlaf. (The therapy of schizophrenia. Insulin shock, cardiazol, narcosis therapy.) 238 pp.

Therapy of Schizophrenia.—For anyone who is interested in the subject the report of this international meeting is indispensable. A great number of hospitals in various countries which gained experience on the subject are represented, with detailed accounts. Apart from this, the physiological, experimental, neurological, and neuropathological aspects of the problem are dealt with in many papers. Details cannot be abstracted here; the general impression obtained of the therapeutical results of the insulin and cardiazol therapy is an optimistic one.

Vol. 40. No. 1. 1937.

Psychiatrische Untersuchungen über die Folgen der operativen Sterilisierung der Frau durch partielle Tubenresektion. (Psychiatric investigations into the results of sterilization of women by partial resection of the tubes.) H. Binder. 1.

*Über das Adie'sche Syndrome. (Adie's syndrome.) M. Dressler and H. Wagner. 50.

Vergleichend-anatomische Untersuchungen über die basale optische Wurzel, die Commissura transversa Gudden and über eine Verbindung der Netzhaut mit dem vegetativen Gebiet im Hypothalamus durch eine "dorsale hypothalamische Wurzel" des Nervus opticus bei Amnioten. (Comparative anatomy of the basal optic root, the transverse commissure of Gudden, and a connection between the retina and the hypothalamic nuclei ("dorsal hypothalamic root") in amniotes.) E. Frey. 69.

Der Liquor cerebrospinalis bei Syphilis. (The cerebrospinal fluid in syphilis.) V. Kafka. 127.

*Über das Koma und Reflexanomalien bei der Insulinschocktherapie der Schizophrenie. (Coma and abnormal reflexes during shock therapy in schizophrenia.) W. Liepmann. 133.

*Über die Schizophrenie im Kindesalter. (Schizophrenia in childhood.) J. Lutz. 141.

Syndrome pariétal avec mouvements forcés complexes et hallucinations visuelles. (Parietal syndrome with forced complex movements and visual hallucinations.) G. de Morsier and R. Broccard. 164.

Versuch einer Abgrenzung und Bestimmung des Instinktbegriffes. (The definition of "instinct.") M. Nachmansohn. 173.

*Beiträge zur Wirkung der Krampftherapie der Schizophrenie. (The effect of epileptic seizures on schizophrenia.) J. Nyirö. 180.

*Über das Verhalten der Bauchdecken- und Cremasterreflexe bei abdominalen Narben. (The abdominal and cremasteric reflexes in patients with abdominal scars.) A. Pronin. 197.

*Über zwei Fälle von Wilson'sche Krankheit bei einem Geschwisterpaar. (Two cases of Wilson's disease affecting sister and brother.) H. Schwyn. 221.

Adie's Syndrome.—The disease is not a pathological entity, but a syndrome which may be produced by various infectious and toxic causes. Syphilis may also be the cause, but very rarely. The difference between the pupillary disturbance in Adie's syndrome and that of other conditions (specially Argyll-Robertson's symptom) is pointed out in extenso. (K. S.)

Coma and Abnormal Reflexes in Schizophrenia.—The beginning of the coma is indicated by the loss of corneal reflexes and light reflexes. During the shock, in the precomatose and comatose stages, about half of the patients showed brisk tendon reflexes and pyramidal symptoms. Mendel-Bechterew's and Rossolimo's phenomena, however, were never observed. During the stage of restlessness the grasp reflex could be observed. (K. S.)

Schizophrenia during Childhood.—Continuation and conclusion of a previously abstracted paper. The conclusions are drawn from the histories of 20 psychiatric children (16 boys and 4 girls). In 10 children the psychosis started before, in 10 cases after the age of 6. In all cases it started before the age of 10 years. The symptomatology is essentially the same as in schizophrenia of adults. Hallucinations were only observed in patients older than 6 years. Katatonic symptoms are rare. Differential diagnosis and prognosis are discussed. Insulin therapy is recommended with certain limitations. Occupational therapy is also recommended. (K. S.)

Epileptic Seizures and Schizophrenia.—11·7 per cent. of the patients of the hospital (taken from a material of 1,000 cases) could take up work spontaneously, without insulin or cardiazol treatment. From 24 patients treated with cardiazol 12 could go back to their professional work again. (K. S.)

Abdominal and Cremasteric Reflexes.—138 patients were examined. During an initial phase (1–3 weeks) after an abdominal operation the abdominal reflexes were usually entirely absent. In cases of old

scars they may be absent or diminished locally (e.g. the right inferior reflex after appendectomy); a complete absence of the abdominal reflexes, however, was never found in such a case. (K. S.)

Two Cases of Wilson's Disease.—The clinical and anatomical study of both cases shows a similarity in symptoms and course. In both cases mental and emotional symptoms dominated at the beginning, so that psychiatric diagnosis of hysteria and schizophrenia were first made. Both developed later a syndrome of Parkinsonism, similar to post-encephalitic states. (K. S.)

ZEITSCHRIFT FÜR DIE GESAMTE NEUROLOGIE UND PSYCHIATRIE

Vol. 160. Parts 1 and 2. October 1937.

*Über erbliche Zwischenhirnsyndrome und ihre entwicklungs-physiologischen Grundlagen (Dargestellt am Modell des Bardet-Biedlschen Syndroms). (Hereditary between-brain syndromes and their developmental physiological background exemplified by the Bardet-Bield syndrome.) F. Panse. 1.
*Die Sippschaft der Mongoloiden Idiotie (Kinship in mongolian idiocy). H. Schröder. 73.
*Über die Endangiitis obliterans (v. Winiwarter-Buergersche Krankheit) unter besonderer Berücksichtigung der Hirnveränderungen. (Endangiitis obliterans (v. Winiwarter-Buerger disease) with a consideration of the brain changes.) E. Sträussler, R. Friedmann, and J. Scheinker. 153.
Zur Frage der Körperbaubestimmung bei Geisteskranken. (Bodily types in the mentally ill.) Dim. T. Dimitrijević. 179.
Psychiatrische Bestandesaufnahme im Gebiet eines medizinisch-anthropologischen Zensus in der Nähe von Rosenheim. (The occurrence of psychiatric disorders, as found in a medical anthropological census in the neighbourhood of Rosenheim.) C. Brugger. 189.
*Wernickesche Krankheit bei chronischer Gastritis. Ein Beitrag zu den Beziehungen zwischen Magen und Gehirn. (Wernicke's disease in chronic gastritis: a study of the relations between stomach and brain.) K. Neubürger. 208.
Multiple Sklerose und Erbanlage. (The hereditary basis of multiple sclerosis.) F. Curtius and H. Speer. 226.
Das vegetative Nervensystem im Verlaufe der Schizophrenie. (Dynamismus der vegetativen Störungen.) (The vegetative system in the course of schizophrenia and the dynamics of the vegetative disturbances.) E. V. Maslow. 246.
Zur Pathologie der optischen Agnosien mit verhältnismässig geringer Lesestörung. (The pathology of optic agnosia with slight disturbance of reading.) O. Pötzl. 255.

Hereditary Between-Brain Syndromes.—A study of two families showing in one three children with the syndrome better known as the Laurence-Moon-Biedl syndrome and in the other one example of the syndrome with one mongol. A full analysis of previously reported cases is given. The hereditary syndrome with polymorphous manifestations may be regarded as due to a fixed gene combination. The physiology of development is under the control of an "organization" centre in the diencephalon (or the precursor of such a centre). The constitutional type is fixed at an early stage. Similarly, the constitutional type of such conditions as tuberous sclerosis, von Recklinghausen disease, and mongolian idiocy are fixed by a hereditary diencephalic disturbance of development. (G. M. G.)

Kinship in Mongolian Idiocy.—Various views of the ætiology of mongolian idiocy are discussed, such as the exhaustion theory, age of mother, smallness of amniotic sac, and changes in the uterine mucosa from the use of chemical birth control methods. The author has tabulated the kindred of 50 mongols in an attempt to find a hereditary factor. He finds a certain degree of evidence in favour of a recessive hereditary factor from the relative preponderance of minor physical abnormalities and feeblemindedness in the families studied. (G. M. G.)

Endarteritis Obliterans.—A study of the cerebral lesions in a man showing the characteristic changes of endarteritis obliterans in cerebral and somatic arteries. His mother had a similar condition verified at autopsy. The brain findings consist largely of scattered scars, often radial, in the distribution of the smallest vessels in the cortex and white matter. The vessels show an obliterating endarteritis with no evidence of inflammatory reaction. The pathology of the syndrome is discussed with especial reference to inflammatory and non-inflammatory lesions. The question of temporary obliteration by spasm is considered in view of the variability and temporary nature of many of the symptoms. (G. M. G.)

Wernicke's Disease in Chronic Gastritis.—Two cases of chronic gastritis with no history of alcoholism are reported. In both multiple punctate hæmorrhages with areas of encephalitis were found, especially in the mamillary body, posterior colliculus, and medial geniculate. Lesions were also found in the thalamus, olives, and dentate nucleus. The lesions are compared with those found in alcoholic and in carcinogenic Wernicke's disease. Various ætiological theories are discussed, bacterial toxin, achylia (the condition is not often found in pernicious anæmia), endogenous autointoxication involving the brain, the question of liver function, and vitamin deficiency. The author considers that in his cases the condition arose acutely shortly before death. (G. M. G.)

Vol. 160. Part 3. November 1937.

Über ein subependymares, multiples, malignes Glioblastom. (A subependymal, multiple malignant glioblastoma.) F. Kino. 297.

*Die umschriebene Atrophie der Basalganglien. (Circumscribed atrophy of the basal ganglia.) F. Bonfiglio. 306.

Über intravenöse PH-messungen in experimentellem epileptischem Anfall (durch Cardiazol). (Intravenous PH measurement in experimental (cardiazol) epileptic fits.) H. Selbach. 334.

Die serologische Tuberkulosediagnostik in Neurologie und Psychiatrie. (Serological diagnosis of tubercle in neurology and psychiatry.) K. F. Scheid. 346.

*Die Neurosecretion des Sympathicus. (Neurosecretion in the sympathetic system.) R. Gaupp. 357.

*Zur Frage der pathologischen Anatomie der Schizophrenie. (The pathological anatomy in schizophrenia.) G. Peters. 361.

Erbbiologische Untersuchungen an Kindern von Epileptikern. (Investigation of the heredity of children of epileptics.) G. Franke. 381.

Spasmus und Rigor. Eine vergleichende physiologische Studie. (Spasm and rigor, a comparative physiological study.) H. Altenburger. 402.

Über ein parietales Störungssyndrom. (A parietal-disorder syndrome.) R. Klein. 417.

Verfall der Gesittung bei Hirngeschwülsten. (Deterioration of morals in brain tumours.) M. de Crinis. 426.

Eine neue Modifikation der Herstellung der bei Liquoruntersuchungen verwendeten Goldsollösung. (A new modification of the preparation of the gold sol solution used in C.S.F. investigations.) L. v. Unghváry. 441.

*Todesfall infolge Insulinshockbehandlung bei einem Schizophrenen. (Klinische und pathologisch-anatomische Beschreibung.) (Death from insulin shock treatment of schizophrenia, a clinical and pathological-anatomical study.) R. Leppien and G. Peters. 444.

Circumscribed Atrophy of the Basal Ganglia.—This reports the case of a man with a long illness beginning at 35 with excitement and megalomania. After 4 years this changed to mental dullness with palilalia and palikinesia and a temporary dumb period and later dysgraphia. Physical deterioration preceded death. There were never any motor or extrapyramidal symptoms or aphasia. Autopsy revealed a general atrophy of both hemispheres with gross atrophy of the head of the caudate nucleus. There was parenchymal degeneration of the head of the caudate and the anterior part of the putamen and pallidum, with destruction of the large cells. There was subependymal gliosis in the caudate nucleus. The author discusses other basal ganglion syndromes and the relationship with Pick's disease of this case, but considers that it does not fall into any of these categories. (G. M. G.)

Neurosecretion in the Sympathetic System.—The authors find in some cells of the superior cervical ganglion in man colloid droplets similar to those described in the hypothalamic nuclei. This is considered to be evidence of secretory activity in the sympathetic nerve cells. (G. M. G.)

Pathological Anatomy of Schizophrenia.—Various areas of the cortex in 14 cases of schizophrenia and in eight hanged people (of normal mentality) were studied by the Nissl technique. In both groups scattered areas of cell outfall and cell alteration without glial reaction were found. The author discusses points of technique which account for these appearances, and considers that the cell alterations are agonal and have nothing to do with the pathology of schizophrenia. (G. M. G.)

Death from Insulin Shock Treatment of Schizophrenia.—A young schizophrenic was treated with seven injections of insulin. He developed unilateral convulsive movements, then clonic spasms, stupor, and a rise of temperature, dying four days later. The brain showed much congestion with some extravasation. There were perivascular glial masses in the white matter, especially occipital, also in the medulla. In the cortex was diffuse ganglion cell change, especially of the large cells and to a less degree in the striate, dentate, and olive. There was change in Sommer's sector and in some Purkinje cells. The lesions are considered to be toxic in nature. (G. M. G.)

Vol. 160. Parts 4 and 5. January 1938.

Der erbliche Muskelschwund. Genealogische Untersuchungen bei neurospinaler Muskelatrophie. (Hereditary muscular atrophy, genealogical research in neurospinal muscular atrophy.) H. Boeters. 455.

Der Aufbau des Gedächtnisses und das Problem der Erinnerungslücken. (The building up of memory and the problem of failure of memory.) E. Roenau. 511.

Studien über den Vitamin C-Gehalt im Liquor cerebrospinalis. III Mitteilung. Untersuchungen uber den Vitamin C-Gehalt im Liquor bei Hungerversuchen. (Studies in the Vitamin C content of C.S.F. Part III. Investigation of the Vitamin C content of the C.S.F. in hunger experiments.) M. Kasahara, T. Kasahara, and M. Horie. 528.

*Zur Symptomatologie und Diagnose der Hirncysticerkose. (The symptomatology and diagnosis of cerebral cysticercosis.) J. Rothfeld. 530.

*Das Synapsenproblem im Sympathicus. (Versuch einer morphologisch-physiologischen Betrachtung.) (The problem of the synapse in the sympathetic system (a morphological and physiological consideration).) E. Herzog and B. Günther. 550.

Ein Beitrag zur Huntingtonschen Erkrankung. (A study of Huntington's disease.) H. C. Hempel. 563.

Untersuchungen zur Beleuchtung der Intoxikationstheorie bei der Dementia Præcox mit besonderer Berücksichtigung der Versuche mit Totaltransfusionen. (Researches towards the elucidation of the intoxication theory of dementia præcox, with special reference to experiments in whole blood transfusion.) P. J. Reiter. 598.

*Genetisch verschiedene Gruppen entwicklungsgestörter Gehirne. (A generic grouping of malformed brains.) H. Jacob. 615.

*Beitrag zur Krampfbehandlung der Schizophrenie. (A contribution on the convulsive treatment of schizophrenia.) R. Stähli and O. Briner. 649.

Über Beeinflussungswahn und Charakter. (Delusions of "influence" and character.) L. Sachs. 680.

Das Körperbauspektrum. (The "spectrum" of bodily build.) W. Plattner. 703.

Untersuchungen über den Fett- und Lipoidabbau in anämischen Nekroseherden bei einem Spätfall von amaurotischer Idiotie. (Investigation of the fat

and lipoid in anæmic necrotic areas in a case of late anaurotic idiocy.) G. Friedrich. 713.
Basedowsche Krankheit und Handschrift. (Handwriting in Grave's disease.) H. Unger. 728.
*Zur Insulinshockbehandlung der Schizophrenie. (Insulin shock treatment of schizophrenia.) P. Plattner and E. Frölicher. 735.
Die Psychopathologie des Cervantes. Ein Versuch ihrer Darstellung auf kulturgeschichtlicher Grundlage. (The psychopathology of Cervantes: a study of his works on the basis of a story of a culture.) W. Leschmann. 767.
Zur Dynamik der Wahnideen. (Auf Grund von Beobachtungen in Fällen von "schizophrenen" Psychosen bei postencephalitischem Parkinsonismus.) (The dynamics of delusional ideas (Observations on cases of "schizophrenic" psychoses in postencephalitic Parkinsonism).) A. Erb. 793.
Kurze methodologische Bemerkung zu meinen Arbeiten über die genetische Bedingtheit der Homosexualität. (Short methodological remarks on my work on the genetic theory of homosexuality.) T. Lang. 804.
Tertiär-syphilitische Veränderungen bei Neurolues. (Tertiary syphilitic lesions in neurosyphilis.) F. Knigge. 810.

Brain Cysticercosis.—Four cases are described. In one cysticercus was diagnosed from membrane found in the C.S.F., the symptoms were meningitic. In one migraine symptoms were found to be due to cysticercus in the choroid plexus and consequent hydrocephalus. In the third symptoms were of a cerebellar tumour. In the fourth epilepsy occurred in a patient known to have had a tape worm. The presence of eosinophil cells in the C.S.F. is discussed as a diagnostic point in the absence of skin or muscle cysticercosis. The value of the precipitation test of Trawinski in the blood is discussed. (G. M. G.)

The Synapse in the Sympathetic System.—Staining of a sympathetic ganglion with a silver impregnation method shows that the neurones are individual, there is no fibrillary continuity or syncytium. The passage of impulses is complicated and not explained by one mode of action. There is a biological oscillation, in the first place electrical, then associated with chemical and colloidal changes. (G. M. G.)

Generic Grouping of Brain Maldevelopments.—The author groups certain developmental abnormalities of the brain according to the time of development when such abnormalities arose: (1) a general arrest of neuronal migration causing symmetrical heterotopias; (2) circumscribed migration arrest causing scattered heterotopias: these occur at a very early stage; (3) micropolygyria and pachygyria (cases of verrucose polygyria described in detail). This is an arrest of development of the fibre system, as distinct from glial, secondary, or pathological microgyria or "ulegyria." (G. M. G.)

Convulsion Therapy of Schizophrenia.—The author gives detailed analysis of 112 cases treated by cardiazol. All types and duration of case were treated. During the course of treatment there were several subluxations, two fractured scapulæ, several cases with considerable rise of blood pressure, and two cardiac arrhythmias. Two cases died, one from embolus from a thrombosed pelvic vein and the other from bilateral hypernephroma. The best remissions occurred in the group of acute hyperkinetic catatonic cases and in the group of late presenile cases. The numbers in each group were rather small for statistics. (G. M. G.)

Insulin Shock Treatment of Schizophrenia.—The author gives details of the method and results of treatment in 67 cases. The results were not as good as those of the Vienna clinic. Of 16 recent cases there were 10 good remissions; of nine up to 1½ years only one remission; of 15 old but relapsing type, 5 remissions; and of 27 old chronic type no remissions. Those of a psychogenic type gave better results than those of a manic-depressive type. The cases giving the best results were under two months' duration. Those with good remissions had 3·2 per cent. of the comas and 1·6 per cent. of the insulin convulsions, much fewer than the failures had. (G. M. G.)

Vol. 161. Parts 1 and 2. March 1938.

Bericht uber die dritte Jahresversammlung der Gesellschaft deutscher Neurologen und Psychiater in München vom 20–22 Sept. 1937. (Report of the third annual meeting of the Society of German Neurologists and Psychiatrists, Munich, 20th–22nd September, 1937.)

This volume contains the papers read at this meeting. Most of them are reviews of the subject and not suitable for abstracting, or short papers abstracts in themselves.

Klinik der Hirngeschwülste. (Clinical aspects of brain tumours.) H. Pette. 10.
Über Hirngeschwülste. (Brain tumours.) O. Gagel. 69.
Über Hirngeschwülste. (Brain tumours.) W. Tönnis. 114.
Cerebrale Zirkulationsstörungen bei Hirngeschwülsten. (Disturbances of cerebral circulation by brain tumours.) G. Bodechtel and G. Döring. 166.
Operative Erfahrungen bei Hirntumoren. (Operative experiences in brain tumours.) F.-W. Kroll. 188.

These are the main reviews on brain tumour from various aspects. Gagel gives a review on the pathology of tumours; Tönnis gives a surgical viewpoint on diagnosis, localization, treatment, and prognosis. There are several short papers dealing with other aspects of the subject.

Epilepsie Klinischer Teil. (Epilepsy, clinical aspect.) K. Pohlisch. 267.
Epilepsie, Vererbung und Konstitution. (Epilepsy, heredity and constitution.) K. Conrad. 280.
Epilepsie Anatomischer Teil. (Epilepsy, anatomical aspect.) A. v. Braunmühl. 292.
Epilepsie, Pathogenese und Therapie. (Epilepsy, pathogenesis and treatment.) K. H. Stauder. 321.

These papers give general reviews on the subject of epilepsy without adding to knowledge.

*Gehirnbefunde bei Christian-Schüllerscher Krankheit und allgemeinen Cholesterinosen. (Brain findings in Christian-Schüller disease and generalized cholesterinosis.) J. Hallervorden. 384.
Das Experiment in der Psychotherapie. (Experiment in Psychotherapie. W. Enke. 444.
Das Experiment in der Berufsberatung. (Experiment in vocational guidance.) O. Graf. 458.

Brain Findings in Christian-Schüller Disease.—This is a review of published cases of the disorder which had changes in the central nervous system. The lesions generally consist of a patchy, ill-defined demyelinization with preservation of axis cylinders and some gliosis rather like disseminated sclerosis. There were granular cells containing greenish pigment with thionin, a brownish-yellow with fat stain, and giving the iron reaction. In some cases there appear to be well defined firm areas with collagen and glial fibres containing foam cells staining strongly with Sudan. Inside these areas vessels are few, but the periphery is rich in vessels. It appears that in some cases there are more or less obvious brain changes with lipoid granulomata in the organs, while in others there are obvious diffuse lipoid changes in the brain. The condition is primarily one of lipoid defect with differences in quantity and localization. (G. M. G.)

ZENTRALBLATT FÜR NEUROCHIRURGIE

Vol. 3. No. 1. 1938.

*Zur Operation der Meningiome der Siebbeinplatte. (An operation for meningioma of the ethmoidal region.) W. Tönnis. 1.
*Über die Behandlung des metastatischen Gehirntumoren. (The treatment of secondary tumours of the brain.) J. H. Zaaijer. 7.
*Untersuchungen und Beobachtungen über die Enstehung vorübergender postoperativer Herdsymptome und postoperativer Liquorveränderungen. (Investigations and observations on transient post-operative focal symptoms and post-operative changes in cerebrospinal fluid.) P. Röttgen, H. Selbach, F. G. v. Stockert, and W. Tönnis. 12.

Meningioma of the Ethmoidal Region.—Tönnis describes an operation for removal of the meningiomas of the olfactory groove, which avoids resection of one or the other of the frontal lobes. A transverse incision is made and after the skin flap has been turned down forwards, the frontal bone is removed widely in one piece, to be replaced at the end of the operation as a free graft. The dura is divided transversely low down the superior longitudinal sinus having been first secured. The frontal lobes are now separated and gently elevated, when the tumour comes into view. The lower anterior, attachment of the falx is of course cut through. The tumour is now dealt with in the usual way. Tönnis has successfully removed three olfactory groove tumours in this way. He mentions that Olivecrona has also removed three meningiomas by this route. (Julian Taylor was probably the first to use this method.) (G. J.)

Secondary Tumours of the Brain.—Zaaijer reports 37 cases with metastatic tumours operated upon in the Vienna clinic of Schonbauer during the last eight years. In 25 cases the tumour was solitary. In 27 cases radical methods were employed; in the others decompressions only. Only six patients lived longer than one year in good physical condition. In three cases where there was metastasis from an undiscovered primary the patients lived 33, 51, and 76 months after removal of the secondary tumour. Zaaijer believes that radical operation is better than decompression, and should be carried out if the metastasis is known to be solitary. (G. J.)

Transient Post-operative Focal Symptoms and Changes in C.S.F.—This is an important co-ordinated piece of work on the effects of operation for tumour removal on the patient. The authors report aphasic disturbances after left-sided hemispheral operations, regardless of the site of the tumour. They ascribe the effects to cerebral œdema, at its height on the third or fourth post-operative day. The disturbance coresponds with the Gerstmann type (finger agnosia, right-left disorientation, arithmetical failures, aphasia). Sensory aphasia was found only as a direct result of tumour pressure and in no other cases. The psychic disturbances are not produced by the direct trauma of operations; they vary from delirium to severe disturbances of consciousness and are attributable to arachnoiditis, cerebral swelling, and œdema. On the biochemical side there seems to be a slight drop in the C.S.F. alkali reserve, amounting to 20 vols. per cent., and at the same time an increase in protein content. The mastix curve indicates an irritative meningitis as being present. (G. J.)

Vol. 3. No. 2. 1938.

Diskordantes Auftreten von Hirntumoren bei erbgleichen Zwilligen. (The occurrence of cerebral tumours in one of identical twins.) O. Petersen and H. Geyer. 53.
*Weitere Mitteilungen über Klinik und Histologie des Kongenitalen arterio-venösen Aneurysmas des Gehirns. (Further contributions on the symptomatology and histology of the congenital arterio-venous aneurysm of the brain.) W. Sorgo. 64.

Venöses Angiomder Dura. (Venous angioma of the dura of the brain.) P. Röttgen. 87.
*Subarachnoidalcysten des Gehirns. (Subarachnoid cysts of the brain.) M. Zehnder. 100.
Extracerebrale Arachnoidalcyste der linken Grosshirnhemisphare. (Extracerebral arachnoidal cyst of the left cerebral hemisphere.) G. Okonek. 112
Über die Hirnschwellung bei Grosshirn geschwülsten. (Cerebral œdema in brain tumours.) G. Häussler. 119.
Fedor Krause und Neurochirurgie. (Fedor Krause and neurosurgery.) C. M. Behrand. 122.
Die Vasomotorik der Gehirndurchblutung. (The vasomotor mechanism of the cerebral circulation.) D. Schneider. 127.

Congenital Arterio-venous Aneurysm of the Brain.—Sorgo reports nine cases from Tönnis' clinic of congenital arterio-venous aneurysms, of which two were cured by tying the afferent vessel and two by total extirpation. In the other cases the carotid artery was tied. The important cases in this series are the two in which the tumour was excised. Both were young soldiers. In the first case the patient fell doing a march and lost consciousness for ten days. On recovering he exhibited weakness of the left arm and leg. A few months later he began to have epileptic attacks in the left arm and leg. It was thought that he had an aneurysm and arteriography was carried out. This showed an arterio-venous aneurysm in the right pre-motor area, high up. This angeioma was removed at operation. In the second case papillœdema was present. Cistern puncture was done with recovery of red cerebrospinal fluid. Air was injected and a large air-filled cyst was discovered in the right occipital lobe. Arteriography demonstrated an aneurysm, the size of a cherry, in the same situation. At operation the meninges were infiltrated with blood and on opening the cyst the aneurysm was found and removed. Only one case is fully described in which the contributary vessels were closed with silver clips after opening up the fissure of Sylvius and tracing the vessels to the tumour. The fistulous point was afterwards found. The furrow between the second and third frontal convolutions was also opened. When the vessel within was clipped the pulsation stopped. This was a left-sided operation and aphasia followed but did not persist. (G. J.)

Subarachnoid Cysts of the Brain.—Zehnder describes eight cases of subarachnoidal cysts (two inter-hemispheral, three of the fissure of Sylvius and three in the cisterna cerebello-medullaris). Their nature is not very clear, but some at least seem to be congenital. Zehnder suggests that some may result from cerebral œdema producing adhesions in the subarachnoidal space. These cases are important because although others have met with them and seme have been described, more information is required concerning them. (G. J.)

BOOK REVIEWS

A TEXT-BOOK OF MENTAL DEFICIENCY

A. F. Tredgold

(6th Edition. Baillière, Tindall and Cox, London. 1937. 25*s*.)

The newest edition of this standard and authoritative work is evidence of the increasing interest taken in all that bears on the social aspects of deficiency. Thus extensive changes have been made in the chapters on ætiology, with especial reference to heredity, and that on sociology, in which the number and fertility of defectives, illegitimacy, venereal disease, crime and pauperism among them, their employability, and finally methods of prevention by sterilization and otherwise, are discussed. Many other alterations have been made in the book, to keep it in line with recent research. The chapter on pathology, however, is still unsatisfactory, undue weight being given to unconfirmed or antiquated work.

THE OCCUPATIONAL TREATMENT OF MENTAL ILLNESS

John Ivison Russell

(Baillière, Tindall and Cox, London. 1937. 6*s*.)

As a practical manual this work is excellent. The account of the theoretical and clinical problems involved is unsatisfactory, but as the work is written primarily not for physicians but for mental hospital nurses, upon whom the carrying out of occupational treatment mainly falls, this is not a grave defect. The presentation is easy to follow, as well as likely to arouse the enthusiasm without which occupational therapy cannot succeed. In the first half of the book the organization and daily practice of the treatment is outlined, with some clinical descriptions, and in the remainder (compiled with the help of craftsmen in the hospital of which Dr. Russell is Superintendent) the procedure in various useful occupations is concisely but adequately explained.

PETER KÜRTEN: A STUDY IN SADISM

George Godwin

(The Acorn Press, London. 1938. 5*s*.)

Any narrative of the astonishing career of Peter Kürten, the so-called " Monster of Düsseldorf," cannot fail to evoke general interest. Psychologists too have their special reasons for tracing the mental processes which led to the diversity and magnitude of this murderer's record, and he is at times held up as the outstanding instance of a sadist at war with society. The interplay of the factors of heredity with those engendered by deplorable mishandling in childhood, and later by twenty-five years of incarceration, add to the complexity and interest of the case. Medico-legal practitioners will also be intrigued by certain of the aspects, and especially by the problem of Kürten's responsibility in law. Nevertheless, it is not easy to determine why this little study was published or for what class of reader it is

intended. Little or no documentation is given of the criminal story, and the psychopathology is treated only sketchily. The author has obviously borrowed heavily from the material contained in Professor Berg's exhaustive article in the *Deut. Ztschr. f.d. ges. Gerichtl. Med.* for 1931.

It is unsatisfactory to find even in a pseudo-scientific work evidences of errors in fact, and the mis-spelling of proper names does not serve as a recommendation. Apparently this essay was originally intended as a preface to an English version of the history of Peter Kürten which is to appear later from the pen of Professor Berg himself. Those who are interested in medical slants on criminology will await this latter monograph with lively anticipation.

PAPERS ON PSYCHO-ANALYSIS

E. Jones

(Baillière, Tindall and Cox, London. 1938. 25*s.*)

The fourth edition of Dr. Jones's book contains 32 articles published previously in various journals. They are classified into general papers, papers on dreams, clinical papers, and papers on child study. In each section the papers are arranged roughly in historical order ; in spite of many footnotes and postscripts, a number of the papers are mainly of historical interest. The author rightly anticipates the reader's question : " Why the paper on recent advances in psychoanalysis (1920) is not followed by another on more recent advances." The reader would have been the more interested, because Dr. Jones tells him that such a paper could not have been a simple summary of Professor Freud's later writings, since some of the most recent advances are still under discussion.

As it stands, the book does not attempt to give an account of the whole field of psychoanalysis. The subjects chosen are dealt with interestingly. The articles are comprehensible even to the non-sectarian, though he may have difficulty sometimes in accepting some symbols or interpretations as " evident." Of great interest to the psychiatrist are Dr. Jones's views on the Future of Psychoanalysis, and on Psychoanalysis and Psychiatry. In the article dealing with the future he says that he " scarcely anticipates much help from such fields as anthropology, political economy, or sociology. . . . More perhaps may be expected from biochemistry and especially from comparative psychology."

The paper on Psychoanalysis and Psychiatry contains among other points a theory on the interaction of toxic and psychological factors in organic illnesses. The views about the origin of " mental morbidity " developed therein are certainly not much different from the " multidimensional " diagnosis aimed at by modern psychiatrists. The glossary will be welcome to both medical and psychological readers. A complete index adds to the value of the book.

MANUAL OF PSYCHIATRY AND MENTAL HYGIENE

Aaron J. Rosanoff, M.D.

(7th Edition. Chapman and Hall, London. 1938. 37*s.* 6*d.*)

This standard text-book has been largely rewritten, and the lack of incongruities between the old and new material is evidence of the task having been well done. An effective plan of division of the book, especially from the standpoint of the student, is adhered to. The first portion provides a general orientation in regard to ætiology, symptomatology, and neuropsychiatric syndromes in the broader sense, as well as some consideration of organic pathology. This is followed by a detailed consideration of particular neuropsychiatric conditions, grouped according to a classification of the

author's, based in so far as possible on ætiology. Space is fairly apportioned between the organic and functional disorders, but in regard to the latter a rather unorthodox grouping is adhered to. Manic depressive insanity is accorded a place, but one looks in vain for the usual headings of schizophrenia and the psychoneuroses, ultimately to find them rather indiscriminately presented under the title of " chaotic sexuality." Here, also, the author partly abandons his eclecticism and shows a marked leaning toward psychoanalytic concepts. Therapy is well if rather briefly presented, and the chapter dealing with psychoanalytic technique is more than ordinarily palatable. Mental hygiene is considered from a very broad aspect, and includes the problems of eugenics, sociology, control of venereal disease, and drug addiction. A brief discussion of psychogenic disturbances in children is given.

A chapter on special diagnostic procedures is well worth the space. A glossary of psychiatric terminology is included in the general index, a practice which does not enhance the value of either. An adequate bibliography follows each chapter. The book embodies most of the recent advances in the field, but omission of any mention of electroencephalography and a scant page devoted to the shock treatment of schizophrenia seem rather lamentable in a text-book bearing the date 1938 on its title page.

ESSENTIALS OF PSYCHIATRY

George W. Henry

(3rd Edition. Baillière, Tindall and Cox, London. 1938. 22*s.* 6*d.*)

This book is unequal in its value. It is helpful to the student in those parts in which the author gives mere descriptions of the various diseases or where he deals with practical questions, such as psychiatric nursing, a chapter which is usually neglected in most of the common text-books. In these chapters the author's great clinical experience shows itself. Where the reader is introduced to general psychopathology, however, matters are often simplified in an astonishing way. In dealing with psychotic phenomena too much stress is laid on psychological interpretations which are derived from psychoanalysis, individual psychology, and analytical psychology. In the chapter on " Personality Disorders " the impression is obtained that practically all psychotic symptoms may be interpreted by these psychological methods. " In those disorders characterized by delusions the affected person obtains protection from the recognition of personal inferiorities, failures, and discrepancies by transferring the responsibility from them to others. . . . It appears in such conditions " (catatonic states !) " that the struggle between the social and more primitive desires is so keen that a disorganization of the personality has resulted. . . . Hysteria, a third form of psychoneurosis, usually has its origin in forbidden sexual experiences which may be real or fantasied and which occur in childhood." The introduction of some recent physiological and biochemical investigations would have done much to enhance these chapters. When reference is made to the physiology of the nervous system it is surprising how definite the information is about the function of the basal ganglia and their influence on personality disorders. It is with these chapters that the critic finds himself in disagreement with the author, and it is unfortunate that besides many valuable chapters several are given over to a simplification of neurophysiology rather than to an introduction to the elements of physiology.

THE ADRENAL CORTEX AND INTERSEXUALITY

L. R. Broster *et alia*

(Chapman and Hall, London. 1938. 15*s*.)

240 pages are given to a study of this subject. The most valuable part of the book is that given to a short description of many cases. A few more details would have enhanced the value of some of the reports. There follows a series of studies of the psychological disturbances found in such cases. Included is a psychological study of homosexuals without glandular disturbance, of eunochoids, and of an eunuch. Though good, the interpolation of this material is confusing, for the title of the book indicates that the authors intend to deal with dysfunction of the adrenals. The pathological study which follows would have been made much more valuable by the inclusion of some photographs of histological material. This could have been accomplished without additional expense, as the clinical photographs in the earlier chapter are excessive in number, many being quite redundant. The biochemical studies are of interest and will stimulate further work. The book suffers from too much repetition and the inclusion of material not necessarily associated with dysfunction of the adrenals.